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THE WORK AND AIMS OF THE UNITED STATES PUBLIC HEALTH SERVICE

THOMAS PARRAN, M.D.*

WASHINGTON, DISTRICT OF COLUMBIA

THE beginning of the United States Public Health Service was an act of Congress signed by President John Adams in 1798. Under the provisions of this act, twenty cents a month was deducted from the wages of each seaman on the merchant ships of the United States, the total deposited with the Collector of Customs, and used to build and operate a series of marine hospitals giving medical care to sick seamen in the principal ports. From this disconnected system of hospitals, supported by a compulsory health-insurance law, there evolved a national Marine Hospital Service. As time went on, provision was made in these hospitals for the care of other federal beneficiaries. Doctors treating seamen in the marine hospitals came into intimate contact with exotic diseases, acquiring proficiency in the diagnosis of yellow fever, of cholera and, later, of bubonic plague and typhus fever. When these diseases appeared in the ports and elsewhere the Marine Hospital Service was called upon for assistance, which increased in value when foreign quarantine also became a function of the Service, the original state-operated quarantine stations having been found lacking in uniformity and efficiency.

Federal help for local authorities during epidemics of foreign origin led naturally into co-operation for control of other epidemic diseases. Following the epoch-making discoveries of disease causation, the first research in preventive medicine by the Service was set up in 1901 by the establishment of the Hygienic Laboratory, now the National Institute of Health; and the Public Health Service acquired its present name on its one hundred and twentieth birthday in 1912.

Compared with the traditions of clinical medicine, the very concept of public health is new. Before Father Pasteur we treated disease, but were without scientific bases for preventing it. Experiences of the last generation, however, seem to prove that control of disease for the population as a whole can be accomplished only by integrating prevention and treatment and following close in the wake of ever-advancing knowledge. This will be even more true in the future as medical science gives us increasing knowledge concerning the prevention and cure of diseases against which our weapons are not now effective.

Additional responsibilities having been laid upon the Public Health Service through successive acts of Congress, our work now can be described broadly under three headings:

1. The giving of medical care to seamen, to the coast-guard personnel, to federal prisoners, to Indians and to other legally named beneficiaries.
2. The application of existing medical knowledge for the control of disease through the voluntary co-operation of state and local health authorities and through guarding against disease from foreign sources.
3. The search for new knowledge concerning causes of disease and better methods for its prevention and control.

Our best minds are constantly at work to extend the boundaries of such knowledge and to co-operate with others in the task; at the present time we have a working partnership on various research tasks with more than twenty universities and scientific institutions.

There are sound sociologic and economic as well as scientific reasons for more aggressive attention to the nation's health. We seem now to have reached a stage in the evolution of citizenship when all the people, rich and poor alike, are beginning to demand at least the minimum of health protection as a right co-equal with that to liberty and the pursuit of happiness. Freedom from preventable disease is beginning to assume an importance in the minds of many citizens which is comparable to their feelings about freedom of speech, of faith, of assembly, of franchise. By the union of effort on the part of the practic-

*Surgeon General, United States Public Health Service.

Address given at the annual meeting of the Massachusetts Medical Society, Boston, May 31, 1938.

ing physicians, the medical schools and the public-health forces, we have a real opportunity to rescue more citizens from the descending spiral of disease, dependency, delinquency and death. For however much the economists may disagree about other matters, there seems no disagreement as to the fact that we pay for disease whether we cure it or not. And there is no disagreement among the most dispassionate of our scientists that by such a united effort it is perfectly possible to prevent and to cure a large part of the disease burden we accept as inevitable.

Within the past three years a good start has been made toward the development of a national health program. Through the Social Security Act there has been established a continuous policy of assisting states and localities to provide better health service for themselves. For the Public Health Service this has been reasonably easy, because since 1880 we have been working with the states in the prevention and control of disease. The social-security appropriations, which last year amounted to $13,200,000, are expended through the Service and the Children's Bureau for life-saving projects in which the states retain full responsibility, subject only to minimum standards for personnel and performance which have been agreed upon beforehand by the state health officers with the two federal agencies involved. With rare exceptions, states have not attempted to shift their health burdens to Washington. On the contrary, the total federal appropriations represent only about 10 per cent of the cost of public-health work, although the national government pays half the cost of such welfare measures as old-age pensions and of such end results of disease as the care of those blind because of syphilis.

Present programs are reasonably efficient so far as concerns the majority of the plagues of the last generation, such as smallpox, scarlet fever, diphtheria and typhoid fever. Precautions must be taken to hold them in check, but in spite of the fact that until very recently they have occupied most of the attention of health officers, they are no longer among the leading causes of disease and death. On the other hand, where pioneer effort is involved, where new services must be established and new methods of control worked out, there is greater need for federal leadership and assistance.

The control of cancer is an example. Without a single dissenting vote, Congress last year passed an act creating the National Cancer Institute as a permanent part of the Public Health Service and providing not only for research into the causes, treatment and control of cancer, but also for the training of specialists, grants-in-aid to scientific institutions and the purchase and loan of radium.

A significant feature of this work to date has been the confidence in federal leadership demonstrated by the many scientific agencies engaged in cancer study and which, for the first time, have voluntarily offered co-ordination of effort for the common cause. Moreover, the National Cancer Council has been able to gain the service of a greater number of leaders in the many and diverse fields of cancer research than could conceivably have been commanded by any single private undertaking. This, to my mind, presents a highly desirable pattern of federal activity in a health problem. The Public Health Service is in a position to promote, assist and advise state and local effort and, in turn, the federal policy is shaped and directed by the leaders of voluntary effort. The same pattern is being followed in our National Advisory Committee on Pneumonia Control.

Venereal disease is another case in point. On May 25 the President signed the La Follette-Bulwinkle Bill, which authorizes long-time support for federal leadership in the control of syphilis and gonorrhea. In many states not even the foundations of such control work had been laid until the social-security appropriations became available, but this must be spread thin to cover all the health needs of the states. It is utterly inadequate to achieve the intensive effort and the concentrated, continuous drive which twenty years of study and experiment in this and other countries have shown will be required to stamp out syphilis as a public-health problem.

We have far to go before we shall have taken up the lag for preventive medicine between what we know and what we do. In addition to cancer, pneumonia and venereal disease, which I have mentioned, every physician in this audience realizes the possibilities that await the better application of our knowledge to tuberculosis, to heart disease, to infant and maternal mortality and to improving the nutritional status of the population.

Perhaps the greatest pride of the Public Health Service lies in the contributions made to science by our distinguished men, living and dead. What Carter did in the pioneer work which paved the way for Reed's discovery of the mosquito transmission of yellow fever, what Goldberger did on pellagra, Stiles on hookworm, Francis on tularemia, Maxcy and Dyer on endemic typhus, Spencer on spotted fever, Armstrong on encephalitis and psittacosis, and many others in diverse fields, we hope may be matched and surpassed in the future by the workers at the bench in the National Institute of Health and in our far-flung field studies.

I cannot even catalogue our laboratories here. Each is a story in itself. I can only say that in research, as in the application of knowledge, in comparison with the whole job to be done the Public Health Service itself will play a relatively small part. In line with our precedents, however, we expect to do intelligent teamwork with doctors, with official agencies and with scientific institutions, to whom the main objective is the greatest gain in the health of our people.

As for myself, the present spokesman of the Service, I am less interested in its size than in the brains it represents. I dream of a day when, through the help of the Service, in a national effort, there shall no longer be the wide gap between medical science at work and the proof of that science; when doctors doing public-health work everywhere in this country shall be as free from political interference as they are in the career of the Service; when doctors in health services — national, state and local — and all other doctors of the nation, working together, shall have achieved for citizens the new American freedom from preventable disease, from unnecessary disablement and from premature death.

TOLERANCE TO AND TOXICITY OF INSULIN

I. With Spontaneous Eating

FREDERICK M. ALLEN, M.D.*

WITH THE ASSISTANCE OF JAMES H. RICE

NEW YORK CITY

A PAPER on artificial hyperinsulinism formerly presented by the present author[1] before the American Physiological Society was published only in abstract. Many subsequent experiments were performed before a clear interpretation seemed to be reached, and these results like many others, were lost, with the dissolution of the Physiatric Institute at Morristown, New Jersey. As the author has been barred from academic and adequate laboratory opportunities, a disproportionate time has been required for collecting the data to be offered here.

The literature covering the physiological, pathological and clinical effects of insulin in excess may be outlined briefly as follows.

The experience of clinicians from Byworth[2] (1928) to the present has shown that large doses, amounting to 1000 units or more per day, can when necessary be given in diabetic coma without harm and with benefit. Also from the earliest writers (Hetényi,[3] Räihä,[4] Reinwein[5]) to the present (Joslin[6]) there have been reports of danger or death from insulin in cases of circulatory disease. Animal experiments revealed numerous effects, such as functional impairment of the heart (Edwards and Page[7]), changes in blood composition (Briggs et al.[8]), early onset of rigor mortis (Hoet and Marks[9]), and the role of the bulbar centers in convulsions (Olmsted and Logan[10]). In all such work the factor of hypoglycemia was involved.

Aftereffects of high or prolonged insulin administration were found in the form of spontaneous or alimentary hyperglycemia (Macleod,[11] Boller and Überrack[12] and others). The symptoms resulting from insulin were divided into those due to hypoglycemia and those due to secondary discharge of adrenine (Macleod,[11] Rosenberg,[13] Cori[14]). Acute insulin intoxication was divided by Fischler[15] into two phases, namely effects upon the nervous system manifested by depression, excitement, delirium or convulsions, and effects upon the skeletal muscles manifested by loss of sense of position and later atony. He attributed these results hypothetically to methylglyoxal formed from glucose under the influence of insulin. In the absence of definite statements, his paper appears to deal with hypoglycemic conditions produced by ordinary doses of insulin. Chronic insulin intoxication has been described by Dünner, Ostertag and Thannhauser.[16] After "large" daily insulin doses of unstated amount their dogs died in eight weeks or less with normal blood sugar, glycogen-free livers and glycogen-poor muscles; but their records included hypoglycemic attacks. Kurokawa and Hosoye[17] reported that rats died within a few days under treatment with 1 unit of insulin per day — a result which is not valid for American rats.

The question of the effects of high or prolonged insulin dosage, which has obviously been inadequately investigated, has gained added importance from recent developments.

1. Clinically, aside from the temporary use of large insulin doses in coma, increasing numbers of insulin-resistant diabetic cases are being discovered, in which the

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compulsory insulin requirement is measured in hundreds or even thousands of units daily over long periods.

2. Insulin has come into extensive use for fattening nondiabetic patients with various forms of malnutrition, tuberculosis and other infections, and also as an aid in the treatment of digestive disorders, skin diseases and an extremely wide variety of other ailments, the result being that large numbers of adults and children receive considerable doses over long periods (Allen[18]).

3. In all the above treatments, the attempt is made to avoid hypoglycemia or to check it immediately by giving carbohydrate: but the newest treatment of certain conditions of insanity (Sakel[19] and Dussik and Sakel[20]) demands prolonged severe hypoglycemia, produced by insulin doses as high as necessary, sometimes totaling several hundred units within a brief period. Sakel's hypothesis is that insulin temporarily disables the higher, phylogenetically younger nervous centers, leaving the lower and older ones comparatively unaffected. This effect is assumed to be due partly to hypoglycemia (with which may be connected the disappearance of glycogen and also degenerations demonstrated in the nervous system in connection with the treatment), but Sakel also emphasizes a supposed direct effect of large insulin doses upon intracellular metabolism. Apart from the theoretic importance, there is the practical fact that the treatment is dangerous to the extent of causing occasional deaths. If it can be proved whether or not insulin has an effect independent of hypoglycemia, there will be no difficulty in suggesting means either for producing hypoglycemia with low insulin dosage or for giving massive insulin doses without hypoglycemia and its dangers.

METHODS

Blood-sugar determinations were made by either the Benedict picric method or the Epstein micro modification. Questions of the finest analytic accuracy were not involved; the essential need was to learn at least the approximate gradations of hypoglycemia or hyperglycemia in the quickest and simplest way, as a guide to further treatment. Glycogen analyses by the Pflueger method were performed by Mr. Rice under difficult conditions which precluded the desired number of observations. Thanks are due to Dr. Aaron S. Price of the Polyclinic Hospital laboratory for the preparation and examination of slides from formaldehyde-fixed organs, and also for carmine stains for glycogen after alcohol fixation. It is regretted that studies of the nervous system, with reference both to glycogen changes and possible degenerative or other lesions were impossible.

The insulin used was mostly of the standard commercial brands. In the early part of the work the Eli Lilly Company kindly supplied an insulin solution of extremely high concentration, which permitted small dosage volumes and a correspondingly slight possibility of an influence of preservative material. Administration was in the form of single or repeated large doses given subcutaneously or intravenously. With reference to theories of a predominant action of insulin upon the liver, it was ascertained that, at least, for large doses there was no essential difference between this administration of insulin and intraperitoneal injection when presumably a large portion must be absorbed into the portal circulation.

The successful results of authors[21-28] who have had facilities for the continuous intravenous infusion of insulin, in imitation of the normal pancreatic supply, are enviable. Most of this work pertained to establishing the normal insulin requirement of the organism, which was set by Greeley[28] at 0.0051 to 0.035 units per kilogram per hour in depancreatized dogs. Bouckaert and his collaborators,[21] using continuous intravenous glucose infusions in rabbits, concluded that 6.8 units per kilogram gave a maximum of glucose utilization and that all insulin above this dose was inactive.

The ratios between insulin and glucose obtained by the above methods may be compared with those obtained in diabetic patients by various clinicians. Such ratios have been notoriously variable. They differ with the animal species, with the doses of insulin and glucose, with the methods employed and with various known and unknown clinical conditions, so that they may be regarded at best as nothing more than an approximation for practical convenience under certain selected conditions, not as showing a relation for which any fundamental physiological significance has been demonstrated.

Notice should also be taken of the lack of a constant connection between hypoglycemic symptoms and any fixed level of blood sugar, as shown by John[29] and others for patients who develop symptoms at high glycemic levels, and by still other writers for patients and animals that remain symptom-free with marked hypoglycemia. Furthermore, the work of Sakel[19] has stimulated studies of central nervous lesions found after deaths from insulin in nondiabetics, besides those previously known in diabetics (Joslin[6]); but it has not yet been determined whether such lesions result only from hypoglycemia or whether they may occur in insulin intoxication without hypoglycemia.

Aside from the evident necessity of planning to suit the environmental limitations, the method of discontinuous injections used in the present work can be justified as follows: (1) it is the method in actual clinical use; (2) it permits study of a question not heretofore raised, namely the time and conditions of disposal of a large surplus of insulin in the body. It is only unfortunate that the prolonged intravenous method could not have been added for comparison.

INSULIN IN HUMAN PATIENTS

Brief Observations

After cautious trials with ascending doses in several patients in the Physiatric Institute, a strong,

young diabetic was given subcutaneous injections of 750 and 1000 units of insulin. According to memory (these records being lost), his age was thirty, his weight 155 lb. (70 kg.), and his insulin requirement 75 units per day. Figures 1, 2 and 3 are reproductions of some of the lantern slides shown at the reading of the original paper in

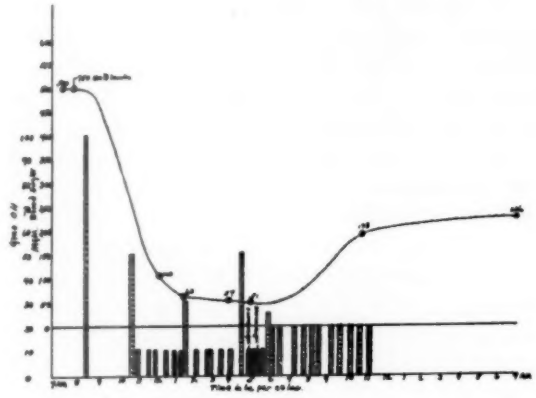

Figure 1.

1932. One point of interest was the size of the doses, which appeared alarming because so much higher than any known to have been given to any patients up to that time, except for those with special conditions such as coma.

These trials, together with animal experiments, disposed of fears that such doses would demand a proportionately huge supply of carbohydrate. Instead of the 100 gm. of starch and sugar, as shown in Figure 1, it was learned that any small amount, such as 10 gm., would act temporarily as an antidote for 750 or 1000 units of insulin but that these feedings had to be repeated, because

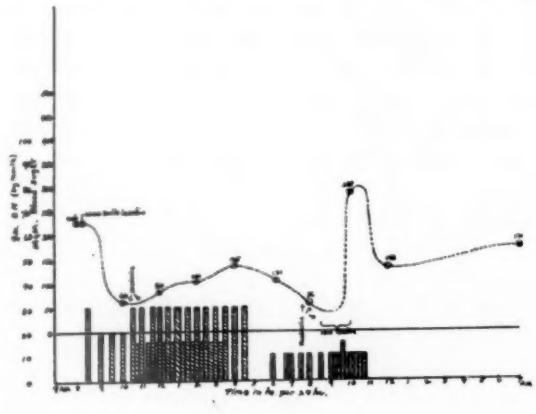

Figure 2.

the patient was unable at any one time to eat enough to give protection through the entire duration of the effects of such a dose. For comparison, Figure 3 shows that protection was afforded by a series of intravenous injections of glucose of only 10 gm. each.

The blood-sugar curves indicate that the effects of the insulin lasted for about twenty-four hours. This duration, that is, the time required for disposing of the surplus insulin, was not appreciably changed by alterations in the amount of carbohydrate used as an antidote or by the high or low level of blood sugar during this time.

The patient experienced hunger and faintness only when there was marked hypoglycemia. Otherwise he felt well. In particular, the absence of disturbance of appetite or digestion at all times appears significant as indicating that these doses

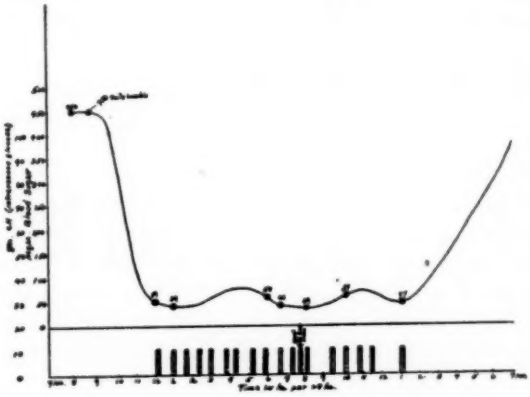

Figure 3.

were well within the bounds of safety, and that they did not establish the maximum limit of insulin tolerance in this individual.

Prolonged Observations

According to the writer's studies, partly published and unpublished, and also those of Falta,[10] Rosenberg,[13] and others, nondiabetic patients with simple malnutrition or mild tuberculosis may be grouped in three classes as regards their response to insulin, as follows:

1. A small number tolerate large doses seemingly indefinitely, so that finally the insulin must be stopped and the diet reduced; otherwise these persons would probably become very obese. The highest dosage in this group was 320 units per day, given to a man with moderately advanced but afebrile tuberculosis, who, receiving 80 units every six hours, had to be fed at two-hour intervals day and night. He thus gained weight with extreme rapidity, without any disturbance attributable to the insulin.

2. The greatest number of patients gained weight with various doses of insulin, without hypoglycemia or any discomfort if they were fed frequently enough, until the weight rose to or slightly above the figure which had been normal to them in health. At this point insulin, in the doses previously well tolerated, began to produce anorexia and malaise. The treatment therefore had to be stopped because of some physiologic reaction which refused to allow the weight to be raised above a definite limit.

3. A small number of patients have proved primarily

intolerant to insulin. These are mostly thin, nervous women with chronically poor appetites and digestions; a few are obese and nervous. They may react badly to doses of 5 units or less, and are often dismissed as unfit for insulin treatment. Nevertheless, by giving doses as low as 1 or 2 units for several weeks, they can often be trained to a point where they finally tolerate larger and larger amounts and gain considerable benefit.

While some of these patients may be easily subject to hypoglycemia, the disturbances mentioned are not obviated by merely raising the blood sugar with any form of carbohydrate administration. With reference to the nervous or hormonal basis, it may be noted that men on the average tolerate considerably more insulin than women; the patients with the highest tolerance are usually phlegmatic, while those with the lowest seem to be invariably nervous, perhaps with some special sympathetic-autonomic relations.

INSULIN IN ANIMALS

Brief Observations

In these experiments also it was necessary to break with the current ideas of insulin-carbohydrate ratios.[30] In view of the definition that 3 units of insulin must reduce the blood sugar of a 2-kg. rabbit, starved for twenty-four hours, to the convulsant level of 45 mg. per cent within five hours, it was surprising to find a rabbit of this kind protected against 25 units of insulin merely by eating a few lettuce or cabbage leaves.

The insulin tolerance of different laboratory species, under conditions of spontaneous eating, is governed chiefly or wholly by the influence of insulin upon appetite. On this basis cats seem to have generally a low tolerance, but owing to their individual variability the idea of establishing a precise limit was abandoned. They will frequently eat after doses of only a few units; increase of appetite was never seen, though these tests were not numerous. A dose that is at all large will cause not only refusal of all food but also the vomiting of whatever may have been eaten with good appetite a short time before. Cats will therefore go into hypoglycemia and die with comparatively small doses of insulin, unless saved by parenteral glucose injections.

Increase of gastric contractions and secretion was demonstrated in dogs by Bulatao and Carlson[31] with 20 to 40 units of insulin, and by La Barre[32] with 20 to 25 units. These effects were connected with definite levels of hypoglycemia and were counteracted by glucose injections. On the other hand, dogs receiving hundreds or thousands of units promptly develop depression, anorexia and vomiting. As they therefore require treatment with glucose parenterally, their records will be given in a subsequent paper. It may be mentioned here that this effect is not due to hypoglycemia, because it appears within the first few minutes, before there is time for much change in either sugar or glycogen in the body, and it is neither prevented nor cured by glucose injections. It would be of interest to have this apparently direct effect of insulin studied by Carlson's instructive method. The effects of the largest doses are very prolonged, being demonstrable for over forty-eight hours as regards both hypoglycemic tendency and other symptoms.

Rabbits have a fairly definite tolerance. They continue to eat with doses of 50 to 75 units, though the eagerness and the quantities eaten are distinctly subnormal. With higher doses, eating is absent or trivial in amount. These statements apply to 2-kg. animals and subcutaneous insulin injections, intravenous injections being much less powerful, as will be shown in subsequent papers. Correlations between insulin and diet can be illustrated. A rabbit receiving 25 units of insulin ate 100 gm. of cabbage within the first six hours, and 200 gm. during the remainder of the twenty-four hours, without symptoms. No rabbit has been able to protect itself against 50 units by eating only greens. But a more concentrated carbohydrate diet can afford protection against doses of 50 to 75 units. For example, a rabbit starved for the usual twenty-four hours, then given 75 units of insulin and fed oats and carrots, ate more slowly and intermittently than a normal hungry animal, but consumed 70 gm. oats and 70 gm. carrots within twelve hours, after which it was able to fast overnight without symptoms. Doses of 100 to 125 units are invariably fatal regardless of any diet, because the animals stop eating. Similarly, with tube feeding a rabbit was protected against 50 units of insulin for twelve hours by receiving 4 gm. of starch and 2.5 gm. of glucose, and during the night had to be given 0.6 gm. starch and 1.5 gm. glucose. A rabbit was kept symptom-free after 75 units of insulin with tube feedings totaling 20 gm. of glucose in 20 per cent solution.

Fasting rats are known to develop convulsions with very small doses of insulin, and fasting mice are so highly sensitive that they have been used for the most delicate biological method of assay. But with feeding, the rat family has the highest insulin tolerance of all known mammals. A 20-gm. mouse fed on bread remained symptom-free with 20 units of insulin, but died with 25 units. The finicky character of the appetite is remarkable; for example, a mouse which readily protects itself against 20 units by eating bread may go into hypoglycemic coma on 2 units rather than eat the crust.

The tolerance of rats is still higher. McIntyre and Burke[33] have reported that rats endure 200 units per kilogram of weight on single days, or 500 units per kilogram in three days. Numerous trials, however, show that their tolerance is higher in proportion as the diet is attractive and rich in carbohydrate. They will protect themselves against very high dosage by merely eating dry glucose or drinking glucose solution. The best diet thus far tried is moist bread, with or without glucose solution. On this, rats weighing from 100 to 250 gm. display a tolerance for 300 to 1000 units of insulin, representing such fantastic figures as 2000 to 4000 units per kilogram. The amounts of carbohydrate required to balance some of the larger doses are shown in Table 1, the animals being selected to illustrate individual variations among a much larger series.

Table 1. *Carbohydrate Balancing Insulin in Rats.*

RAT NO.	WEIGHT	INSULIN, SUBCUTANEOUS	CARBOHYDRATE BY STOMACH	GLUCOSE, SUBCUTANEOUS	DURATION OF TREATMENT	RESULTS
	gm.	*units*		*gm.*	*hr.*	
1	210	200	3.5 gm. glucose	6.0	44	Symptom-free while drinking glucose for 8 hours; then refused it, and subcutaneous injections for remaining 36 hours failed to save life.
2	260	200	6.5 gm. glucose	—	36	Fasting afterward; symptom-free; recovered.
3	250	320	2.5 gm. glucose	2.0	14	Drank glucose (10 per cent solution in 0.85 per cent NaCl solution) in 8 hours; then lost appetite and died after 6 hours of intravenous injections.
4	185	320	2.5 gm. glucose	—	8	Hungry before insulin injection; refused food afterward, but drank glucose very slowly during 8 hours; then fasted; remained symptom-free.
5	220	320	8.5 gm. glucose	3.0	34	Drank glucose to remain symptom-free during 27 hours; then developed hypoglycemic convulsions when glucose was withdrawn; died 7 hours later in spite of subcutaneous injections.
6	240	320	7 gm. cornstarch plus 4 gm. glucose	3.0	28	Did not relish the mixture of boiled starch and glucose; occasional subcutaneous injections totaling 3 gm. were necessary to check hypoglycemic attacks; symptom-free after 28th hour.
7	200	320	40 cc. milk by stomach tube	7.0	40	Refused to drink but showed no indigestion from tube feedings; hypoglycemic convulsions recurring during 40 hours were checked in each instance by prompt glucose injections, but death resulted nevertheless.
8	215	320	25 gm. bread	1.0	39	Symptom-free with eating bread during 26 hours; then food was removed; developed hypoglycemic convulsion after 5 hours, and another 8 hours after that, each cured by 0.5 gm. glucose injection; recovery.
9	300	400	15 gm. bread plus 2.5 gm. glucose	—	15	By alternately eating bread and drinking glucose solution remained symptom-free.
10	390	800	23 gm. bread plus 2.5 gm. glucose	1.4	33	By eating bread and drinking glucose remained symptom-free for 22 hours; then food was removed; and mild hypoglycemic attacks occurred for 11 hours; recovery with small subcutaneous injections.

Individual differences interfere with the fixing of a precise upper tolerance limit. Also, the capricious appetite must be considered, for if there are changes of diet, surroundings or nervous influences, together with maximum insulin doses, there may be refusal of food and consequent death. Under normal conditions, the prodigious insulin tolerance is in harmony with the remarkable general efficiency of the rat as a physiologic machine. Analyses show that the blood sugar commonly falls to levels a little above or below 50 mg. per cent. Other species under these conditions lose appetite with various smaller doses of insulin. The rat is saved by its steady persistence in eating the trifle of bread or drinking the trifle of glucose solution necessary to ward off fatal hypoglycemia. With the highest doses the animals are usually quiet, perhaps from lassitude with low blood sugar, but there is at least the teleological advantage of avoiding increase of sugar consumption by muscular activity.

The maintenance of hyperinsulinism over a considerable time is easily demonstrated. For example, a rat receiving one of the maximum doses may appear thoroughly well and comfortable; but if after twenty-four to thirty-six (or sometimes more) hours food is removed, death may occur in hypoglycemia. The site of storage of the excess insulin is of interest. When insulin is injected above or below the ankle, and amputation is performed at the knee or hip after twelve to twenty-four hours, considerable fluid is found in the area of injection, together with hemorrhagic inflammation in the tissues. The results shown in Table 2 are conflicting. An early cessation of the hypoglycemic tendency, indicating that most of the insulin store is removed with the leg, was found in only a minority of the instances, while in the majority an asthenic hypoglycemic state seemed to persist after the amputation. These findings were probably due to the weakness of these species of animals. Subsequently it became possible to use a species (cats) which has better strength to withstand the surgery and prolonged fasting. In them

the injected fluid becomes absorbed without trace, but the decisive termination of hypoglycemia leaves no doubt that the principal insulin depot is at the site of injection and that any factor of constitutional hyperinsulinism is slight or transitory. This retention at the injection site for twelve or eighteen (and probably more) hours is longer than has heretofore been recognized for insulin.

Gigante[34] cites authors who have required doses ranging from 8 to 500 units of insulin per kilogram in order to produce hypoglycemic convulsions in different species of birds. The differences between species are therefore very great, and the first effect of the large doses is a rapid gain of perhaps 30 or 40 gm. in weight, due to increased eating, as proved by weighing the diet. Later the weight becomes stationary, and finally falls. As other investigators have found that insulin in smaller doses does not increase metabolism, so also here the decline in weight seems to be accounted for merely by reduced eating. By weighed diets it is then learned that a rat can tolerate 100 units of insulin per day, while eating no more bread than it was consuming before the insulin was started. The rats become more and more subject to temporary failure of appetite, requiring

Table 2. *Insulin Injections in Leg, and Subsequent Amputation.*

Animal No.	Weight	Insulin, Subcutaneous	Duration of Treatment		Subcutaneous Glucose		Result
			Before Amputation	After Amputation	Before Amputation	After Amputation	
	gm.	*units*	*hr.*	*hr.*	*gm.*	*gm.*	
Rat 1	125	250	12	2	2.2	0.4	Recovery.
Rat 2	250	320	18	17	1.5 subcutaneous 5.5 by stomach	2.0	Recovery; death 2 days later.
Rat 3	210	320	24	15	4.0	2.5	Progressive weakness; death.
Rat 4	220	320	24	13	4.5	1.5	Weakness; recovery.
Rabbit 1	2000	250	12	7	24.0	7.0	Death from hypoglycemia.
Rabbit 2	2000	250	16	12	16.0	12.0	Progressive weakness; death.
Rabbit 3	2000	250	25	17	16.0	16.0	Progressive weakness; death.

resistance displayed by the pigeon is especially remarkable. Apparently there is resistance both to the lowering of the blood sugar below a certain level, and also to the occurrence of convulsions with low sugar, so that both metabolic and nervous peculiarities may be involved. The most remarkable point is that the above figures apply to starved animals. Even after a ten-day fast involving a 20 per cent weight loss, a pigeon remained symptom-free with 125 units of insulin per kilogram. Many species of birds therefore are entirely out of a class with mammals and cannot be compared with them on any rational basis of insulin tolerance.

Prolonged Observations

The writer's experience seems to agree with the general verdict expressed in the literature (Long and Bischoff;[35] Aubertin and Mollaret[36]) that no great degree of obesity can be produced or maintained by insulin.

In the present series, prolonged experiments were limited to rats. Doses much above 100 units have been tolerated for only a few days. The maximum limits for individual animals in various series have been as follows: 100 units daily for three weeks, 75 units daily for four weeks; 50 units daily for six weeks — all on bread diet. The a glucose injection to save them. Finally the loss of appetite and nutrition reaches a point which entails either death, stopping of the experiment, or entire dependence upon parenteral injections. Examples of the quantities required for protection will be given in a subsequent paper.

SUMMARY AND CONCLUSIONS

Great increases of insulin dosage increase the intensity of effect only slightly, so that very small supplies of carbohydrate suffice for antidoting. Most prominent is the increased duration of effect, so that the requisite carbohydrate cannot possibly be supplied at one time but must be administered over a considerable period. The active influence of a single dose of insulin for twenty-four hours in man or forty-eight hours in animals is longer than previously known. These relations have a bearing upon any attempts to calculate insulin-glucose ratios within any fixed time period.

A state of insulin intoxication is recognized by symptoms of depression, malaise, weakness, anorexia, vomiting and circulatory failure, not due to hypoglycemia. It can be recognized in animals before the occurrence of hypoglycemia, and is not prevented or cured by glucose. It depends upon the production of pathologic hyperinsulinism and is widely independent of the absolute insulin dos-

age, varying between species, individuals and physiologic states. It is not produced by the largest doses that may be required to supply a need or deficiency in diabetic coma or other resistant states. The largest doses ever given to a patient not in such a special state (750 to 1000 units, or ten to fourteen times the daily requirement) failed to produce these symptoms, indicating that the tolerance of many persons must be higher than this. On the other hand, doses of only a few units can produce the typical condition in predisposed persons.

The insulin dosage which can be tolerated without intoxication in the above sense has been determined more or less accurately for several animal species. For a strong man it is probably above 15 units per kilogram of weight. For the dog and cat it appears to be less than this. For the rabbit it is 35 to 40 units per kilogram, for the mouse 1000 units, and for the rat 2000 to 4000 units. For birds, it is still higher.

This series may offer some support for Sakel's hypothesis, in showing lower insulin tolerance of the species having greater development of the higher nervous centers. Also the fact that insulin-sensitive persons are usually nervous while those with higher resistance are more phlegmatic may, if generally confirmed, be significant from the psychiatric as well as the general physiological point of view.

Two broad statements of practical application may be made for the laboratory and clinic respectively: (1) no animal ever succumbs to insulin hypoglycemia while eating up to the capacity of a normal hungry individual of the species — loss of appetite always precedes any dangerous symptoms; (2) the high tolerance of strong or average persons confirms the prevailing view of the nontoxicity of insulin for them up to an extremely high limit, but it does not imply that similar doses can be given safely to weak or sensitive individuals.

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TRAUMATIC RUPTURE OF THE DIAPHRAGM IN A CHILD

Report of a Case

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TRAUMATIC rupture of the diaphragm, which used to be a rarity, bids fair to be much commoner because of the frequency of automobile accidents. This being so, the case here reported may hold a special interest.

CASE REPORT

On August 6, 1937, a boy of 5 was brought into the accident room of the Josiah B. Thomas Hospital, Peabody, Massachusetts, having been knocked down or run over by a truck. The diagnoses were concussion, multiple contusions, lacerations, abrasions and probable internal injuries.

In addition to wounds on the scalp and face, there were extensive abrasions on several parts of the body, into which sand was deeply ground. These areas included the left shoulder and the left anterolateral aspect of the chest. The right posterolateral aspect of the chest showed an extensive contused area. The abdomen was slightly distended and very tender, not only superficially on account of the abrasions but also to deep pressure, and there was moderate involuntary muscle spasm. A circular area of discoloration surrounding the anus was observed, apparently due to extravasation of blood. The condition of shock was pronounced, so much so that very little attempt was made to treat the wounds other than by irrigation and wet dressings. The patient vomited at intervals.

On account of the extensive skin areas which had been injured, the usual physical examination of the chest was impracticable. The condition of the abdomen raised the problem of whether or not it should be opened. As the shock wore off the fact that there was injury to the abdomen became more apparent, but the spasm of the abdominal muscles did not increase during the 24 hours following the accident, and surgical intervention was deferred. The spinal fluid showed the presence of many red blood cells, as did the urine. There was also blood in the vomitus and in the stool. The area of discoloration around the anus increased in size for about 24 hours, then became stationary. Dullness in the left chest was first interpreted as probably due to extravasation of blood. The effects of the concussion wore off in 3 or 4 days, and the superficial wounds responded to antiseptic treatment. The pulse, however, remained rapid and the heart was found to be displaced to the right. After about a week, the abrasions were clean, and several areas were grafted with pin-point grafts.

When, about 3 weeks after admission, the condition of the skin of the chest permitted more careful examination, the physical signs proved most interesting. The area of cardiac dullness was shifted markedly to the right; the apex beat was visible to the right of the sternum. In the left chest there were large areas of flatness and dullness and some of hypertympany. The dullness was not constant in certain areas but was replaced by hypertympany at times. Auscultation revealed in the left chest tinkling characteristic of gas in the bowel. The abdomen was scaphoid. The patient complained of pain in the left chest and the left upper quadrant of the abdomen. His appetite was poor, and he vomited occasionally after eating. X-ray examination revealed loops of the bowel in the chest and displacement of the mediastinal contents to the right (Fig. 1). A barium enema demonstrated that the colon was in the left pleural cavity (Fig. 2), and a small barium meal showed the stomach to be in the chest also. It was completely inverted (Fig. 3).

Figure 1. *Gas in loops of bowel in the chest and increased density in the region of the left lower lobe. The left leaf of the diaphragm cannot be identified.*

Inasmuch as the patient was not constantly vomiting and showed no signs of obstruction, it was decided to defer operation until a maximum amount of gain in physical condition had been obtained. The patient was given careful supportive treatment and improved markedly. When, however, about 4 weeks after the accident, vomiting became a more prominent feature and the boy seemed to be losing a little ground rather than gaining, further delay seemed to be unjustified. Preliminary crushing of the left phrenic nerve was done and the patient was transfused twice.

At operation on September 15 intratracheal anesthesia was administered, cyclopropane being used almost entirely, with a little ether at one stage of the operation. I believe that this procedure was among those which did most to permit successful surgery. The skin incision followed the left costal margin rather closely. The left rectus muscle was split from its insertion almost to the umbilicus, where its fibers were cut transversely. Exposure

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was found to be excellent through this incision. As soon as the abdomen was entered and explored, a small tube was passed into the stomach by the anesthetist, to serve as a landmark and to deflate the stomach.

Most of the large bowel was found to be in the chest; even part of the ascending colon was above the diaphragm, indicating that this part of the colon had a mesentery, a not uncommon finding. The appendix was not far from the opening in the diaphragm. Practically all the small bowel was also in the chest. About half the spleen was above the diaphragm, as was the left lobe of the liver. The

Figure 2. *Lateral view showing the course of the barium enema extending into the chest.*

stomach, of course, was entirely in the chest. The opening in the diaphragm was very large, about 10 cm. long and 5 cm. wide. It was almost exclusively in the central portion of the left leaf, although the tear extended across into the right leaf for about 2 cm. The diaphragm was flaccid and easy to manipulate. There was no peritoneal covering or sac for the eviscerated organs which were found in the chest. There were adhesions of various degrees of firmness; most of them were tenuous, but a few were very strongly adherent, requiring sharp dissection. The firmest points of adhesion were along the edge of the opening in the diaphragm, where presumably the raw surface caused by the rupture was favorable to fibroblastic proliferation. There was, however, no significant hemorrhage connected with the separation of any of the adhesions, and the viscera were readily removed from the chest cavity. The abdominal cavity could not hold all its normal viscera while the wound was open, but they were kept warm and moist with packs while the diaphragm was being repaired.

This repair consisted of two rows of linen mattress sutures with an overlap of from 0.6 to 2 cm. Two sutures were placed to the right of the esophagus, with a small overlap. The mattress sutures were held without tying until all had been placed. A very small catheter was run into the chest alongside of the esophagus while the abdomen was being closed, in an effort to equalize the pressure between the thoracic cavity and the abdominal cavity. The anesthetist used moderate positive pressure to expand the lung after the diaphragm was closed, but no attempt was made to expand it completely. The abdominal wall was closed in layers. The catheter running into the chest was left in place until the last stitches in the abdominal wall were about to be tied.

A transfusion of 250 cc. of blood was given postoperatively and the patient was put in an oxygen tent. He was in considerable shock. The mediastinal contents' being still displaced to the right, the possibility of positive pressure in the left pleural cavity was considered, in spite of the measures taken to avoid this complication, so that about 4 hours after operation a paracentesis needle was inserted in the left chest under local anesthesia. The pressure, however, was found to be slightly negative. There was no postoperative vomiting, and the patient had a comparatively smooth recovery. Intravenous glucose solution, 10 per cent, was administered for 4 days, 1000 cc. a day. X-ray pictures were taken intermittently, the first postoperatively on the day of operation (Fig. 4). A few days later a

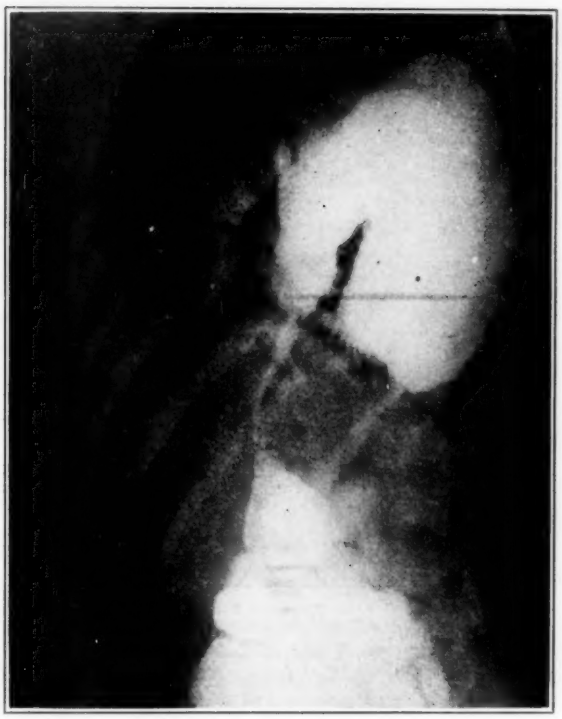

Figure 3. *Oblique view showing complete inversion of the stomach in the chest, and some barium in the large and small bowel in the chest.*

small amount of fluid was observed to have accumulated in the left pleural space, but it was absorbed spontaneously. The left lung expanded steadily, and the mediastinal contents gradually assumed their normal positions. A small zone of inflammation which developed in the right lung shortly after the operation disappeared in a few days.

The patient was discharged from the hospital October 13, 4 weeks after operation. Physical examination at this time showed the chest and abdomen to be essentially normal, and an x-ray film confirmed the finding. Figure 5 shows an x-ray picture of the chest taken at discharge, and

Figure 6 one of the stomach and duodenum after a barium meal.

Since leaving the hospital the patient has gained strength and weight. On December 15 he weighed 45 lb. and was well and active, whereas he had weighed 30 lb. in the hospital before operation. Examination on December 24 showed the wound to be strong and the general condition to be good. On April 15, 1938, the boy's family reported him to be still well.

I had examined this boy a few months before the accident, going over his chest for evidence of respiratory infection, and had found none of the abnormal signs indicative of displacement of the thoracic organs. The dullness, flatness, tinkling, and so forth characterizing the chest findings after the accident would have been difficult to miss had they been present. It is evident that the abnormal situation of the abdominal organs did not exist prior to the accident. Furthermore, in June, 1937, the patient had had a complete examination by another local doctor who reported that the heart and lungs were normal.

Figure 4. *Picture taken four hours postoperatively showing the displacement of the mediastinal contents to the right and the collapsed left lung. Note the great empty space in the left pleural cavity and low position of the left leaf of the diaphragm, due to tension caused by repair. Paracentesis demonstrated that there was no positive pressure in the left pleural cavity at the time this picture was taken.*

Harrington, who reported[1] on the surgical treatment of 105 cases of diaphragmatic hernia, wrote, in an earlier article,[2] regarding the traumatic type: "From a practical standpoint, I believe that all

Figure 5. *Picture taken a month after operation. The mediastinal contents have resumed their normal position. The left lung is completely expanded. The fluid which had accumulated in the left pleural cavity has been entirely absorbed. The left leaf of the diaphragm has resumed its dome-like shape.*

Figure 6. *Immediate and sixteen-hour barium meals taken five weeks postoperatively. The stomach and duodenum are normal, and the position of the colon is approximately normal. There is no evidence of obstruction.*

cases in which the onset of symptoms follows injury should be placed in this group regardless of whether the injury to the diaphragm was due to direct or to indirect force." In the case under discussion, the *symptoms* of pain in the left chest and left upper quadrant of the abdomen had their onset after the accident, and the *signs* of tachycardia, deep tenderness in the abdomen and blood in the vomitus, sputum, feces and urine testified to a degree of internal injury consistent with a violent disturbance of the organs of the chest and abdomen. The type of injury, as evidenced by the very extensive areas of contusion and abrasion on the walls of the chest and abdomen, as well as the manner of injury, seems to be consistent with traumatic etiology of the pleural evisceration of the abdominal organs. The very fact that there was a large extravasation of blood around the anus is indicative of a degree of force tending to push the contents of the abdomen not only up into the chest but also down through the pelvic floor. The gradual change in the abdomen from being somewhat distended and tense immediately following the accident to being distinctly scaphoid several days later is in itself prima facie evidence that the abdominal viscera were for the most part below the diaphragm on admission and that they subsequently shifted into the chest.

SUMMARY

A case of pleural evisceration of the abdominal organs is presented, with description of the technic of operation through an abdominal approach. Comment upon the likelihood of trauma as the etiology of the rupture of the diaphragm is included and notation is made of a sign — perianal ecchymosis — indicative of extreme intra-abdominal force.

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HEREDITARY ARTHRODYSPLASIA ASSOCIATED WITH DYSTROPHY OF THE NAILS

Report of a Case

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THIS title has been borrowed from a paper by Turner[1] of Atlanta, Georgia, who reported a similar condition in a series of cases in 1933. My case fits his description and classification so well that no other title is adequate.

CASE REPORT

J. B., a 65-year-old printer, entered the Cambridge Hospital as the result of increasing general weakness and chronic digestive disturbance. Dr. Horace P. Stevens found at an exploratory operation that the patient had an inoperable carcinoma of the pancreas. I was asked to see him because of some peculiar condition of his knees.

Examination showed that there were no patellas where one would expect to find them.[2] The knees were broad. The internal condyle was more prominent than usual, and the intracondylar notch could be easily palpated. The patellas were small and were located on the external aspect of both knees. The quadriceps tendon was small, and the muscle no larger than a fingerbreadth. The motions in the knees were practically normal, actively and passively, but active extension was much diminished in power. It was also noted that there was marked limitation of supination of the hands, associated with limitation of extension and flexion of both elbows. The nails on each thumb were rudimentary, and only about 0.3 cm. long. The other nails appeared normal. The soft part of each thumb beyond the end of the nail was broad and soft and tended to be folded back on itself. The patient had never been inconvenienced by the lack of normal motion in the elbows or by lack of normal supination. His legs had bothered him in walking, and he had had difficulty in walking fast or going up and down stairs, owing to his weak and mechanically inefficient quadriceps. His work compelled him to stand all day.

Two nieces of his wife and a nephew had rudimentary nails on all fingers, but so far as the patient knew their knees were normal. On his mother's side, a niece and his uncle also had rudimentary nails on all fingers. He knew nothing about their knees. The patient was a native of Canada and had been away from his relatives many years so that, apart from these observations, he knew nothing about their other hereditary characteristics.

As this condition was obviously rare and unusual, at least in my experience, I carefully searched the literature for similar case reports.

The most complete report is that of Turner,[1] previously mentioned, who presented a study of two families in which 35 of 79 persons were affected with arthrodysplasia and dystrophy of the nails. He states that such a condition had never previously been reported, but he apparently overlooked several previous papers on the subject. These papers were, however, incomplete, and although describing essentially similar conditions were listed under congenital absence of the patellas,

From the Orthopedic Clinic, Cambridge Hospital, Cambridge, Massachusetts.

Read at a meeting of the Boston Orthopedic Club, March 14, 1938.

*Assistant professor of orthopedic surgery, Harvard Medical School; associate surgeon, Cambridge Hospital.

Figure 1. *Photograph of knees from the front showing the enlarged internal condyles with lateral displacement of the patellas.*

Figure 2. *Photograph of knees flexed, showing the enlarged internal condyles. Note the lateral displacement of the patellas.*

and so were misleading. Turner describes, however, the various skeletal defects of the hips, knees, ankles, elbows and shoulders occurring in his case, which are consistent with those shown in

Figure 3. *X-ray photograph of knees in the antero-posterior plane, showing the lateral position of the patellas, with a newly formed articular facet on the lateral condyle of the femur.*

mine. He discusses the dystrophy of the nails and states:

> It varies from almost total absence of the nail to a nail that is scarcely distinguishable from a small nail except that it is thinner. The thumbs are usually

Figure 4. *Lateral views of both elbows, showing various exostoses about the joint. These exostoses limit motion and may be characteristic of any joint with this condition.*

> most affected, the condition becoming gradually less marked across the hand toward the little finger. The typical dystrophic nail starts as a small, normal-appearing nail that is, however, much thinner than normal and that gradually disappears about halfway down the nail bed, allowing the fleshy end of the finger to turn back-

ward over the nail. When the extreme condition is present it appears that the nail has been removed from the digit and not reproduced.

Hereditary arthrodysplasia is apparently observed only in association with hereditary dystrophy of the nails. It is true, however, that the latter may appear quite independently of any joint defect.

Rubin[3] reported 3 cases — those of a mother and 2 children. The mother and 1 child had no thumbnails, as well as absent patellas. One child had absent patellas but the nails were normal. The mother's mother had had no thumbnails. In 1898, Thorndike[4] reported a summary of 51 cases. These varied in characteristics: a few had nail defects, all were apparently hereditary, and many had absent patellas — one or both. The knees were relaxed, and there were many associated congenital defects and deformities. The only criticism offered is that more than half the cases reported were in infants under two years of age. It is generally recognized that the patella often does not develop well until the sixth year, so that observations in regard to its presence or absence before then should not be taken too seriously. Thorndike quoted Little,[5] who, in turn, quoted Sedgwick's note of 18 cases in a family of four generations, in which there were no patellas and no thumbnails. The latter did not state, however, whether the two conditions coexisted in all cases, or whether some patients had one and some the other. In none of the cases presented by Thorndike was any note made of absent or defective nails.

The condition is purely inherited and congenital, and is apparently due to some defect in the closing or fusing of the ectodermal and mesodermal layers of the embryo. The degrees of the deformity vary in different cases; one may show the skeletal defects, another simply nail defects, or the two may be combined, as in the present case. There is no treatment except in youth when it is directed only toward the dislocation of the patellas.

321 Dartmouth Street.

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THE TREATMENT OF PULMONARY TUBERCULOSIS

A Comparison of Home and Sanatorium Methods

RALPH VOLK, M.D.*

BOSTON

THIS discussion of the treatment of pulmonary tuberculosis in the home and in the sanatorium was prompted first by observation of many patients under care during six years of residency in two sanatoriums, and secondly by the opinions of several phthisiologists whom I subsequently questioned concerning these two methods of treatment. This presentation does not include all cases of pulmonary tuberculosis, as is pointed out below.

It occurred to me while doing institutional work that many patients remained in the sanatorium long after they had received the maximum benefit from hospital treatment. These were particularly the chronic fibrocavitary cases, in which the emphysema, secondary chronic bronchitis or bronchiectasis was too extensive or the patient too old to permit surgical collapse therapy. Some patients had been in the sanatorium for from five to fifteen years with no apparent benefit. There are many other patients who apparently can be taken care of at home with as great success as in the sanatorium, providing the home conditions are suitable.

The financial status of the patient is of course of the utmost importance, but this discussion concerns only those patients who can afford to pay small or moderate physicians' fees, although I believe that the fees should be commensurate with ability to pay. Obviously the poor cannot afford prolonged private treatment, nor are their home conditions suitable for it. The wealthy present no problem, for they can afford private sanatorium care, or can have hospital facilities in their own homes if desired.

PRINCIPLES OF THERAPY

The present treatment of tuberculosis has two important and concurrent phases: general and local. Included in general treatment are rest, proper nutrition, adequate ventilation and nursing care. Local treatment consists of collapse therapy in one form or another: pneumothorax, phrenicectomy, thoracoplasty and their refinements.

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Rest can be obtained at home equally as well as in the sanatorium.

Proper nutrition is unquestionably of great importance to the tuberculous patient. He requires a well-balanced diet, adequate in all the food elements, in vitamins and in mineral content. Such a diet can be adequate both at home and in the sanatorium, but in the sanatorium the food is cooked for a few hundred people, many of whom dislike "institutional cooking" and tire of the sameness of the menus. It is impossible to prepare food to suit all the patients. To do this the food must be appetizing, properly seasoned, sufficiently varied and of the patient's choice. I have seen patients miss many meals at the sanatorium because they do not like what was offered them. At home, on the other hand, a patient can usually have the type of food he enjoys, and moreover have it prepared to suit his fancy. The physician can advise the patient's family as to what constitutes a balanced diet.

There should be a proper circulation of air in the patient's room, but it is not necessary to keep it at or near the freezing point, as was so commonly believed at one time. A sanatorium should be well ventilated, or at least should be so constructed that ventilation is adequate. If each patient could have a private room, there would be little difficulty over this problem. Unfortunately, however, where there are two or more people in a room there are frequent arguments, particularly during the cold weather, over whether the steam should be turned on or off and how much the windows should be opened. This argument may seem childish, but it often becomes a real problem in the institution. It is not hard to understand this when we realize that one bed in a room may be in a direct draft, while the other is not. At home, on the other hand, the patient has his own room, and his bed can be arranged in a satisfactory position.

As to climate, I believe it is generally conceded that treatment for tuberculosis is as successful at sea level as at higher altitudes, and equally as successful in the East as in the West.

Nursing care is more nearly satisfactory in the sanatorium than at home. This is particularly true in the case of severely ill patients. However, most cases of pulmonary tuberculosis do not require professional nursing. The necessary care includes only the preparation of meals, the changing of the bedclothes, the administration of the prescribed medication, and a few hygienic measures, discussed below. All these duties can be carried out, in the household, by anyone with ordinary intelligence.

The collapse therapy of choice is artificial pneumothorax. Ordinary intrapleural pneumothorax can be carried out at home without difficulty, and later continued in the physician's office or clinic. With failure to find a free intrapleural space, however, surgical collapse becomes necessary and this requires hospital treatment for at least a short period. With the advent of extrapleural pneumothorax, which will unquestionably replace a large percentage of thoracoplasties, there will be another group of patients who can continue collapse therapy at home following immediate convalescence from the apicolysis.

HYGIENE

Hygienic measures include all procedures aimed to prevent the dissemination of tubercle bacilli.

There is only one way of destroying the tubercle bacilli in sputum: to burn it. Sputum cups can be burned at home as well as in the sanatorium.

In the sanatorium it is impractical to have separate dishes for each patient, whereas at home a few dishes can easily be kept for the one sick person in the house. In the sanatorium, dishes are sterilized, usually properly, in a dishwashing machine, but sometimes the attendant in charge is of inferior intelligence and does not understand the importance of his job. A timing device on the machine, which makes it run for the period necessary for sterilization, obviates this difficulty. Dishes can be boiled at home, and instructions should be given by the physician as to the required time for sterilization.

The chance of becoming infected from handling the patient's bedclothes or from contact with the furniture, walls, doorknobs and doors of his room is slight, and presents no more hazard to relatives and friends at home than they do to the visitors at a sanatorium. Simple hand-washing after contact with contaminated objects is sufficent protection.

In a sanatorium, negative-sputum cases are frequently exposed to open cases, and the noninfected patients object because they fear a setback; it is possible that some reactivations are caused by new exogenous infections. At home the patient is usually careful because he does not want members of his family to become infected.

PUBLIC-HEALTH ASPECT

The boards of health are interested in preventing the spread of the disease and in eradicating it. To accomplish this, programs of case finding are arranged. Besides the services of fieldworkers, diagnostic facilities are made available to private practitioners, and clinics are established. Cases are reported to the boards of health, and these reports

are kept on file. This allows the local board to check up on the patients whenever desirable. Prevention of the spread of tuberculosis does not require segregation in sanatoriums, unless the home conditions in the individual cases are inadequate for proper care of the patient and control of the disease. Furthermore, if more patients can be cared for at home, and if residence is shortened for a large group of sanatorium patients, more funds will be available for case finding.

HOME TREATMENT

Conditions suitable for home treatment can be summarized as follows: a well-ventilated bedroom, to be used only by the patient; an adequate food supply; and some one to prepare and serve the patient's meals, boil his dishes and burn his sputum cups. The home should be located in a district conducive to proper rest, and not where streetcars or elevated trains run late at night.

Under proper conditions, a large group of patients can be cared for at home. Slightly and moderately active as well as quiescent cases of pulmonary tuberculosis, whether classified as minimal, moderately advanced or far advanced, are amenable to home treatment. Artificial pneumothorax cases can receive refills of air at home. Also patients who have had surgery done in the hospital, and who have had an uneventful immediate convalescence, can take late convalescent treatment at home.

SANATORIUM TREATMENT

As already observed, the lack of sufficient funds for private treatment at home necessitates sanatorium care. Where home conditions do not meet the requirements outlined above, the patient should be in a sanatorium. Very sick people with highly active pulmonary tuberculosis who require more than ordinary nursing care obviously belong there, as do patients requiring surgical collapse when intrapleural pneumothorax has failed. Severe coughers and neurotics, who put an undue strain on their families, should also receive institutional care.

PSYCHOLOGIC EFFECTS

In the sanatorium there is a routine which everyone must follow, and rules which are more or less general. This tends to make each patient feel contented in following certain courses of treatment, since he realizes that the other patients must undergo them as well. Frequently, however, I have heard a patient express discouragement because some other patient who "looks no better" than he does, or who has been in the sanatorium a shorter time, is getting more exercise. He feels that either he must be very sick or he is not getting along so well as he should. Seeing another patient have a large or a fatal hemorrhage instills fear into the observer. This may have a beneficial effect, or on the other hand may be very upsetting. A patient who previous to observing hemoptysis in another was not very conscientious about his treatment may become more co-operative, because he fears a similar episode. We expect this reaction, and see it in many patients who were previously hard to handle. On the other hand, the more neurotic patients constantly fear hemorrhage once they have seen another patient with this symptom in a severe form. When one of this group suffers a hemoptysis or even shows blood-streaked sputum, it is extremely difficult to reassure him.

At home, on the other hand, there is no other patient for comparison. He sees no one with a condition he can compare with his own who is getting more exercise than himself. He never witnesses a severe or fatal hemorrhage. If an emergency arises the patient is more readily reassured than is the man who has observed catastrophic results from hemoptyses in others.

REHABILITATION

The rehabilitation of the tuberculous patient who has finished his active treatment is of major importance to everyone, including the doctor, the social worker, the family and the state. A patient who has had more than a brief residence at a sanatorium becomes very selfish concerning his own care and responsibility. Whether he has paid for his hospital treatment or not, he feels obligated to no one. The care that he has had was "coming to him." Either he paid for it or "the state owed it to me, because I paid taxes, too." It is difficult for him to summon enough enthusiasm or ambition to get a part-time job, even if it were available.

An excellent and necessary department in every sanatorium is that devoted to occupational therapy, but it can hardly be expected to prepare the patient for gainful work when he is discharged. Its chief function is to keep patients occupied and happy through creating decorative or useful articles, for their own use or for sale by the sanatorium. Patients cared for at home, on the other hand, usually appreciate the efforts of the family in their behalf. When they improve, they are thankful for the care they have had and are willing and anxious to get back to their jobs.

WHY PATIENTS GO TO SANATORIUMS

If it is true that a large group of patients can be treated as well at home as in the sanatorium,

why are so many patients sent there? Several factors are involved. First of all, most sanatoriums are in the country, and the belief is still prevalent among practitioners of medicine as well as the laity that country air is necessary for curing pulmonary tuberculosis. If the doctor does not suggest that the patient go to a sanatorium, friends and relatives will. Secondly, members of the household may be afraid of the disease, and would rather see the patient admitted to an institution on that account. Thirdly, the family doctor often suggests sanatorium treatment or makes application for admission as soon as he has made the diagnosis. He does this for several reasons: he may be incapable of treating the disease because he is unacquainted with the modern therapy for pulmonary tuberculosis, and it may not occur to him to obtain consultation with a specialist before advising sanatorium treatment. There are very few patients who sincerely desire hospitalization for any length of time, and the physician fails to realize how often it may be avoided.

SUMMARY AND CONCLUSIONS

The important aspects of the prophylaxis and treatment of pulmonary tuberculosis are analyzed, with consideration of the general and local treatment of the disease, the required hygiene for prevention of its spread, and its public-health aspect.

The possibilities of home treatment and the conditions suitable for it are enumerated.

A comparison of the advantages and disadvantages of home and sanatorium residence, including discussion of the psychologic effects of each on the patient and the problem of rehabilitation following treatment, is made.

There are three groups of patients who definitely require sanatorium treatment; those whose financial and home conditions are very poor, the actively sick who require special nursing care, and those requiring surgery.

Other patients with pulmonary tuberculosis can be treated at home under the proper conditions with considerable success.

Most people would rather take treatment at home than in a sanatorium, and should be given this opportunity if conditions are suitable.

The family physician should consult an internist specializing in tuberculosis before advising sanatorium treatment, for it can often be avoided without detriment to the patient. To make this possible, the specialist must limit himself to fees which his patients are able to pay.

311 Commonwealth Avenue.

THE COST OF INSTITUTIONAL CARE OF EPILEPTICS IN MASSACHUSETTS

DOUGLASS V. BROWN, PH.D.,* AND MERRILL MOORE, M.D.†

BOSTON

IT IS the purpose of this paper to present certain estimates of the cost of institutional care of epileptics in Massachusetts. The results are derived as accurately as possible from the data obtainable, but no claim is made to any high degree of precision in the final estimates. They are offered merely as an indication of the magnitude of the costs incurred.

In the preparation of these estimates, certain assumptions have had to be made. These are as follows:

1. It is assumed that the per capita costs of maintenance of epileptics in institutions not exclusively confined to the care of epileptics are equal to the average per capita costs for all patients in these institutions, regardless of the disorders from which they are suffering. Considerable support is lent to this procedure by the fact that costs at the Monson State Hospital, designed exclusively for epileptics, have been over a period of years only slightly lower than the averages for the sixteen institutions from which the basic data are taken.* The fact that the majority of the patients at Monson are classified as "epileptics without psychoses" will probably account for such differences as do exist.†

2. It is assumed that per capita costs in five hospitals§ may be taken as equal to those of the sixteen institutions included in the summary tables prepared by the Department of Mental Diseases. In any case, patients in these hospitals included only about 3 per cent of the resident epileptics on September 30, 1935.

3. Charges for interest, depreciation and costs of central administration have been allocated on the assumption that such charges for resident epileptics bear the same relation to total charges as the number of epileptics bears to the total resident population.

From the Department of Medicine and the Department of Diseases of the Nervous System, Harvard Medical School, and the Neurological Unit, Boston City Hospital.

Read at the annual meeting of the American Psychiatric Association, Section on Convulsive Disorders, Pittsburgh, Pennsylvania, May 10, 1937.

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†Associate in psychiatry, Harvard Medical School.

*Annual Report for Year ending November 30, 1934. P. 120.

†On September 30, 1935, only 6 cases presenting this diagnosis were resident in all the other institutions included in this study.

§The McLean Hospital (private) at Waverley, the mental wards of the Bridgewater State Hospital and of the State Infirmary at Tewksbury and Veterans' Administration Facilities Nos. 105 and 107.

4. It is assumed that the number of epileptics in residence on September 30, 1935 is a fair basis for the calculation of a continuing annual charge. In this connection it is also assumed that the distribution, as of this date, of these patients among the different types of institutions is reasonably typical.

In any interpretation of the results of the study, the limitations imposed by these assumptions should be kept clearly in mind.

* * *

On September 30, 1935, there were resident in the twenty-one Massachusetts institutions considered in this study, 2041 patients whose disorders were diagnosed as epileptic.§ Of these, 1774 were found in the thirteen state hospitals for mental diseases, 200 in three state schools for mental defectives, and 67 in five hospitals, enumerated above. Of the 1841 resident in hospitals, 946 were diagnosed as "epileptics with psychoses."

Per capita costs for maintenance, including medical care, have naturally varied from year to year. There is, however, no discernible periodic trend; fluctuations have tended to follow the cyclical movements of general prices. The average annual per capita cost for the period from 1917 to 1933 was $357.24. In obtaining this figure, receipts from sales of products have been excluded. On the other hand, receipts from paying patients, which in 1934 amounted to about 9 per cent of the total of all maintenance costs in the state hospitals and schools for mental defectives, are included.*

Estimates of capital charges and costs of central administration are available for the years 1917 and 1933.† For the former year, charges for interest, depreciation and central administration amounted to $78.59 per capita of the resident population for that year. For 1933, the corresponding figure is $111.60‡ It may thus be inferred that the trend of overhead expenses has been upward; it is not unreasonable to expect that such a trend is likely to continue. Nevertheless, in order to obtain a conservative estimate of the amount to be added to the costs of maintenance, the average of these two figures, $95.09, has been used in the subsequent calculations.

§No account has been taken of epileptics in other institutions in the State. In this respect, therefore, the estimates presented may be considered as minimal.

*Annual Report of the Commissioner of Mental Diseases of Massachusetts for Year ending November 30, 1934. Pp. 32 and 113.

†Estimates of these items are available only for the two years mentioned in the text. Data on maintenance charges are, however, presented for each year during this period in the annual reports of the Commissioner. See, for example, Annual Report for 1934 (p. 120).

‡The detailed information on which these results are based has been compiled by Dr. Neil A. Dayton, director of the Division of Statistics and Research, Massachusetts Department of Mental Diseases. Where no other source is indicated, the material used in this paper has been prepared under his guidance.

It has not seemed necessary to reproduce the detailed items of capital and administrative charges. For 1933, costs of central administration amounted to less than 12 per cent of all overhead charges.

The sum of the figure for maintenance charges and that for overhead charges, $452.33, may be multiplied by 2041, the number of resident epileptics, to yield an estimate of the continuing annual charge. The result, $923,205, is a minimal approximation of the annual cost of the institutional care of epileptics in Massachusetts. It should be emphasized at this point that no effort has been made to estimate any costs other than those which have actually been incurred in the state institutions included in this study. More specifically, there has been no attempt to ascertain the expenditures in behalf of patients not confined to hospitals or other institutions, nor the loss of income of the patients. The inclusion of these and other items would add substantial amounts to the figures presented. Unfortunately, there are no means available by which even a rough approximation of the magnitude of these other costs may be secured.

* * *

It would be interesting to estimate the total charges incurred in behalf of epileptic individuals over the period of their lives, but in the absence of case histories extending over the entire life of such individuals, this is impossible. Nevertheless, for one small group — those epileptics whose deaths occurred in state institutions — such an estimate may have a limited significance. It cannot be too strongly emphasized that the results are applicable neither to other groups nor to the epileptic population as a whole. It must be remembered, moreover, that the numbers involved are so small as to offer little more than an indication of the probable order of magnitude of the sums involved.

Statistics are available of the number of deaths occurring in state hospitals among epileptics committed by court order, together with the total time spent in institutions during this residence, and, if there had been previous admissions, during these earlier residences as well.* For the year 1934, the number of such deaths was 40; in 1935 it was 44. Of these, 25 and 28, respectively, were first-admission cases; 15 and 16 respectively, had been admitted to state institutions on two or more occasions. The first admissions dying in 1934 had spent, on the average, 8.1 years within institutions; the corresponding figure for those dying in 1935 was 6.6 years. For the readmissions, the average length of stay was 19.5 years for those dying in 1934 and 17.9 for those dying in 1935.

On the basis of these averages, and on the assumption that the annual per capita charges incurred amounted to $452.33, it is possible to obtain rough estimates of the average lifetime costs in-

*See, for example, Annual Report for 1934 (p. 191). Figures for 1935 have been supplied by Dr. Dayton.

curred in behalf of individuals in each of these groups.

Those epileptics who entered a mental hospital for the first time and remained there continuously until their death showed the following average total costs per patient:

First admissions dying in 1934.... $3700
First admissions dying in 1935.... $3000

Epileptics who were in mental hospitals during two or more residences and died during the last of these showed average total costs for all admissions as follows:

Readmissions dying in 1934..... $8800
Readmissions dying in 1935..... $8100

SUMMARY

The annual cost of the institutional care of epileptics in Massachusetts undoubtedly exceeds $900,000, and may be much higher.

Total institutional costs for individual epileptics throughout their lives may mount to $8000, and inasmuch as the estimates given above are based upon averages, to considerably larger amounts in particular cases.

THE NEW HAMPSHIRE MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND FORTY-SEVENTH ANNUAL MEETING

May 17 and 18, 1938

Tuesday Morning, May 17

THE members convened in the Hotel Carpenter, Manchester, at nine-thirty o'clock.

The first part of the morning was devoted to the following round-table conferences:

Medicine. Symptoms and Tests of the Failing Kidney. Leader, Bruce Snow, Manchester.

Surgery. Infections of the Hand. Leader, Clinton R. Mullins, Concord.

Industrial Accidents. Leader, Emery M. Fitch, Claremont.

Orthopedics. The Internist and His Orthopedic Problems. Leader, Fred E. Clow, Wolfeboro.

During the last half of the morning the following round-table conferences were held:

Medicine. The Simplification of Laboratory Diagnosis for the General Practitioner. Leader, Harold D. Levine, Bristol.

Surgery. Sympathetic Surgery in Peripheral Vascular Disease. Leader, M. Dawson Tyson, Hanover.

Obstetrics. Work of Maternity and Infancy Committee. Leader, Benjamin P. Burpee, Manchester.

Dermatology. Treatment of Common Skin Diseases. Leader, J. Harper Blaisdell, Boston.

Tuesday Afternoon, May 17

President Ladd: The first order of business on this afternoon's program is the presentation of the gold medals for fifty-year membership in the Society.

I am very sorry to say that Dr. Alpha H. Harriman, of Laconia, will be unable to be present, but I am going to read a letter from him.

Dear Dr. Metcalf:

I shall not be able to attend the annual meeting. I am very sorry that I cannot go. I think I have attended every meeting of the New Hampshire Medical Society since 1888.

I have some trouble in writing, but I wish you all success in your meeting.

A. H. Harriman, M.D.

Dr. Harriman has given all of us quite a record to emulate; I commend his record to some of the younger men here.

Is Dr. Frederick L. Hawkins, of Meredith, here? Will you kindly step to the platform?

Dr. Hawkins, on behalf of the New Hampshire Medical Society, I congratulate you for your fifty years of service. I am sure that you have done yourself proud and have been a credit to the Society. It is my duty as president, and it is also my pleasure, to present to you this gold medal.

Dr. Hawkins: When I was a young boy, I was associated a great deal with my grandmother who was continually reminding me that children should be seen and not heard. And so I feel the urge to turn backward and be a child again just for the moment!

President Ladd: I am going to ask Dr. Henry O. Smith, of Hudson, to come to the platform.

Dr. Smith, it is my pleasure, as president of this society, to present to you this gold medal which you have well earned. You have been one of our outstanding members, and you have always met the call of your associates. We are very proud of your work and are happy today to present to

you this small token of esteem. We hope you will live to be a hundred!

Dr. Smith: I wish to thank you from the bottom of my heart for this beautiful gift which denotes to me my association with this honorable body for half a century.

Almost all of you here present became members of your state society by joining your county societies, and at the same time you automatically became members of the American Medical Association. That has been true since 1901, when the American Medical Association was reorganized. But prior to then, a person who wished to become a member of the American Medical Association marked his application for such membership in just the way that each one of you applied for membership in your county society.

It so happened that quite early in my career as a physician, I made such application and became a member, as I am now — a fellow of the American Medical Association. And so it happened that although there are now living eight members of the New Hampshire Medical Society who joined it before I did fifty years ago, nevertheless, I am the senior member in New Hampshire of the American Medical Association!

The presentation of the gold medals was followed by a "Symposium on Thoracic Disease," with the following titles and speakers:

Treatment of Pneumonia. Frederick T. Lord, Boston. Discussion opened by Francis H. Nolan, Claremont, and Harold D. Levine,* Bristol.

Cancer of the Lung. Donald S. King, Boston. Discussion opened by M. Dawson Tyson, Hanover.

Empyema. Edward D. Churchill, Boston. Discuscussion opened by William J. P. Dye, Wolfeboro.

The following paper was also read:

Surgical Diseases of the Extrahepatic Bile Ducts. I. S. Ravdin, Philadelphia. Discussion opened by Walter H. Lacey, Keene, and Chester L. Smart, Laconia.

Wednesday Morning, May 18

The first period in the morning, beginning at nine-thirty o'clock, was devoted to the following round-table conferences:

Medicine. Diseases of the Liver. Leader, Leslie B. Copenhaver, North Woodstock.

Surgery. The Acute Abdomen. Leader, Herbert L. Taylor, Portsmouth.

Ear, Nose and Throat. Every-day Problems. Leader, John A. Hunter, Dover.

Pediatrics. Preventive Medicine. Leader, Ursula G. Sanders, Concord.

*Winner of Pray Prize for 1938. Title: "The Opportunity for the Modern Treatment of Lobar Pneumonia in Rural Practice."

At eleven o'clock a general meeting was held in the auditorium.

President Ladd: At this time, we are going to call upon the visiting delegates. The first is from Connecticut, Dr. Paul R. Felt, of Middletown. I understand that Dr. Felt's father was the president of the New Hampshire Medical Society in 1904 and 1905, and it surely pleases all of us to see Dr. Felt, the boy of our ex-president, here today.

Dr. Felt: Now that the question has been brought up, with very kind reference to my good father, I should simply like to say that the boy does not ever expect to be the physician that his father was!

I am very glad to be back in New Hampshire as a delegate from Connecticut. I feel, perhaps, like the proverbial old rat in the proverbial old garret, as my entire life has been exclusively along the lines of psychiatry, and I confess that a lot of these very good papers that have been read here have gone over my head, although I admit that they must be good. But why in the world they chose a psychiatrist to go as a delegate to a general medical meeting, I do not know, unless the others were so busy that they could not go.

President Ladd: The next delegate from Connecticut we claim as one of our own sons. His father was one of the outstanding citizens of this state, and I believe that all of you have at least heard of that grand old man, Dr. Worthen, who served us so faithfully, not only as a professor at Dartmouth College but also as a member of our Public Service Commission. Today, we have his son, Dr. Thacher W. Worthen, of Hartford, with us.

Dr. Worthen: Thank you very much for your kind introduction. It gives me the greatest pleasure to bring the greetings of our society. Having lived in New Hampshire the first twenty-five years of my existence, with the exception of the first five and a half months, I believe I am somewhat of a reliable authority on the State, and it gives me added personal pleasure to be here.

President Ladd: We are now going to hear from the State of Maine. Is Dr. Charles W. Kinghorn, of Kittery, here? [There was no response.] Is Dr. Sam Sparhawk of Burlington, Vermont, here? [There was no response.]

From Massachusetts, we are going to hear from Dr. Daniel J. Ellison of Lowell.

Dr. Ellison: It seems that this is almost an old-home day, because before I left my office, I looked at a couple of pieces of paper in frames on the wall of my office; one was dated August, 1918, when I served on the New Hampshire

Board of Registration in Medicine and the other, dated March 11, 1911, was my membership in the Belknap County Medical Society.

Some years ago, there was a woman lawyer in this state who used to go to Washington at every session of Congress and lobby for woman suffrage. At one of these sessions, she met a representative from Virginia, who said to her: "Madam, I understand that you come from New Hampshire. What do they raise in New Hampshire?" Her answer was: "We raise in New Hampshire everything you do in Virginia, except hell and jackasses!"

Now, there is one thing we do raise in New Hampshire and that is physicians, possibly not great specialists, but able men.

It is really a great pleasure for me to convey to the New Hampshire Medical Society the greetings and the well wishes of the Massachusetts Medical Society.

PRESIDENT LADD: We have another delegate here from Massachusetts, Dr. Henry F. Dearborn, of Lawrence.

DR. DEARBORN: It is a double pleasure to come to your state. I was born in Exeter, and although my family moved to Massachusetts when I was a small boy, I returned here later to go to school. Several generations of my father's family came from this state, and I have always spent my summer holidays on the shores of your beautiful lakes.

It is with sincere pleasure that I bring to you the greetings of the Massachusetts Medical Society.

PRESIDENT LADD: Are there any delegates here from Rhode Island? Is Dr. Auray Fontaine, of Woonsocket, here? [There was no response.] Is Dr. Francis H. Chaffee, of Providence here?

DR. CHAFFEE: It gives me pleasure to extend to you the greetings of the Rhode Island Medical Society.

So far, this meeting has been an "old-home week," as one delegate expressed it. As you may know, Rhode Island is famous for going its own way; it never goes with anyone else. Carrying along that parallel, I must confess that this is only the second time that I have ever been in New Hampshire. However, I must acknowledge that my welcome has been grand. I have had a very good time and shall certainly come back.

PRESIDENT LADD: Dr. H. O. Smith, of Hudson, will now read the financial report of the Board of Trustees. [Dr. Smith then read the following report.]

FINANCIAL REPORT

FOR THE YEAR ENDING MAY 1, 1938

Receipts	
Interest on various bank deposits	$513.00
Expenditures	
Prize awarded to Harold D. Levine	100.00
Increase in funds, other than the Benevolence Fund, in the hands of the Trustees	$413.30
GENERAL FUND	
Deposit: Portsmouth Trust and Guarantee Company	$3,245.49
New Hampshire Savings Bank	5,370.11
Nashua Trust Company	1,569.27
BARTLETT FUND	
Deposit: Portsmouth Savings Bank $6,098.49	
Permanent Fund 352.11	
Accrued income available for general use	5,746.38
Total available for general use — May 1, 1938	$15,931.25
PRAY FUND	
Deposit in the Strafford Savings Bank. $1,000 of this is a permanent fund, the income to be expended only for prize essays	$1,362.31
BURNHAM FUND	
Deposit in the New Hampshire Savings Bank. $1,140 of this is a permanent fund, the income to be expended only for prize essays	$2,083.15
BENEVOLENCE FUND	
Deposit in the New Hampshire Savings Bank. A small final dividend will probably be paid by the Merrimack River Savings Bank, now in liquidation	$1,711.24

A prize of $100 was awarded to Harold D. Levine for an essay entitled, "The Opportunity for the Modern Treatment of Lobar Pneumonia in Rural Practice." It is expected that prizes will be offered in 1938-1939 and notice will be sent to the members of the Society in due time.

The accounts of the Treasurer have been examined and found to be correctly cast and properly vouched.

HENRY O. SMITH,
FREDERIC P. LORD, *Trustees.*

DR. SMITH: I should like to say just a word about the Bartlett Fund. It was given to the Society nearly one hundred years ago by Dr. Josiah Bartlett, Jr., the son of Dr. Josiah Bartlett, the founder of this society.

Dr. Josiah Bartlett, Jr., was president of this society for seven years. He left the sum of $200, which was to remain at interest for ten years, at which time the entire amount was to be considered

a permanent fund, the income of which should be used for the promotion of medical science.

It may be of interest to some of you who may have forgotten or who may never have known the fact that from this fund the Society voted the sum of $1,000 to assist in the re-building of the Dartmouth Medical College.

Dr. Alpha H. Harriman, of Laconia, today concludes a service of fourteen years as a trustee of this society. His colleagues on the Board wish to express their appreciation of his great ability, excellent judgment and spirit of co-operation which has ever been manifested throughout this service that in length of time, we believe, has never been equalled in the history of the Society. We are pleased that the Society has conferred on him the position of trustee emeritus and that this day a telegram has been sent to him conveying the greetings and good wishes of the Society.

PRESIDENT LADD: We will now hear a report from our secretary, Dr. Carleton R. Metcalf, of Concord. But before you hear from him, I want to say a few words.

When I was first a member of this society, about everybody with whom I talked thought and said that our secretary, Dr. Sullivan, was among the best. However, we all realized that he was getting along in years and that the time was approaching when somebody would have to be elected to take his very important position. Everyone seemed to be up in the air at the time of Dr. Sullivan's death. As I have been closely associated with the New Hampshire Medical Society for the last eight or ten years, I knew of Dr. Sullivan's wonderful work. However, as much as we appreciated what Dr. Sullivan did, I want to say, as the outgoing president of this society, that Dr. Metcalf's work has been a real task and that he has done an excellent job; besides that, he is a busy practitioner. And, for the time that he has put in on work for this society and for the devotion that he has shown, I am going to ask that a rising vote of thanks be given to him by the members.

[The audience rose and there was prolonged applause.]

It gives me great pleasure to present to you your secretary, Dr. Metcalf, who will give his report of the House of Delegates.

SECRETARY METCALF: The House of Delegates gave considerable attention to the question of socialization of medicine. Many of you know that there is, at present, a bill before Congress which allows the sum of $50,000 to investigate medicine and the practice of medicine in all its phases, with the thought that recommendations may be made for controlling, regulating or improving medical practice.

The House of Delegates approved of any fair and judicial investigation which will lead to the benefit of the patient or the doctor or both. The House of Delegates does not approve of the plan which was suggested by the Committee of 430, which would subsidize hospitals and subsidize medical schools. The members of the House of Delegates believe that that is a step toward what we are pleased to call state medicine. The House of Delegates instructed its delegate to the House of Delegates of the American Medical Association to take that stand, and they have left the further work in the matter during the ensuing year to the Committee on Public Relations, with power to act.

The House of Delegates approved of the erection of a new Army Medical Library Building in Washington. The Library of the Surgeon General is now very much overcrowded, and a new building is needed.

The Committee on Medical Economics was continued. Dr. Frederic P. Lord, of Hanover, withdrew from the committee, and Dr. Richard W. Robinson, of Laconia, is to take his place.

The Committee for the Control of Cancer was given $60 and the Child Health Committee $50 for printing pamphlets and other literature to be sent to members of the Society.

The Committee on Mental and Social Hygiene was requested to consider the simplification of the present law about the sterilization of mentally deficient persons.

The House of Delegates wishes me to call your attention, and particularly that of the county secretaries, to the Speakers' Bureau. We have a revised list of speakers who are willing to attend meetings and to speak at any county meeting. There are some fifty names on the list, so that the county secretaries and the county societies will have a wide choice.

There was a lengthy discussion about group hospitalization or hospitalization insurance. Two or three years ago, the House of Delegates approved, in principle, of group hospitalization, and they turned the matter over to the Hospital Superintendents' Club for consideration and for action. The Hospital Superintendents' Club is now seriously considering, under the leadership of Mr. Smith, of the Mary Hitchcock Hospital in Hanover, three plans. One plan is to go ahead with group hospitalization in connection with the Massachusetts group, which established group hospitalization several months ago—I believe it was last October. The second plan is to take it up in connection with an accident insurance company in

Vermont. The third plan is to see if it can be taken up better and more reasonably with one of the big life insurance companies, like the Metropolitan or the Travelers.

The policy, which has been tentatively drawn up, is similar to the Massachusetts, New York and New Jersey policies. For the sum of $10 a year, any working man or working woman is allowed not more than twenty-one days' hospitalization in a semi-private room, with the payment of all ordinary hospital care and expenses, except x-ray. Certain diseases are excluded, like contagious, venereal and mental diseases. The members of the family of this working man can get into the plan a little more cheaply. His wife can join for $8 a year, and all the children under nineteen years of age for $6 a year. So that under this plan, each member of the family would be eligible for not more than twenty-one days of hospitalization in any one year, under the above conditions.

The Hospital Superintendents' Club is favorably inclined toward the plan, and probably within the next year it will be inaugurated in several or many of our New Hampshire hospitals. The person who holds the policy may go to any hospital in New Hampshire. If he goes to a hospital outside of New Hampshire, — for example, if he breaks his leg in Detroit and goes to a hospital there, — he will be allowed $6 a day toward his expenses in that hospital up to the twenty-one day period.

The Hospital Superintendents' Club asks the co-operation of this society in making this plan successful, if and when it is put into effect. The House of Delegates has left this matter to the Committee on Public Relations, but seemed unanimously in favor of trying out the plan, if successful arrangements can be made.

One of the doctors in one of our towns was assessed by the selectmen on his drugs and medicines as stock-in-trade. We have an opinion on this matter from our counsel, Mr. Sulloway, who states that it is his opinion that a doctor is not liable for a tax on medicines and drugs as stock-in-trade. I mention this merely to tell you that if any of you run into a similar dilemma, you can probably be successful in defending it.

The next annual meeting of the Society will be held here at the Carpenter Hotel.

The round-table conferences will be continued. We are, however, making a change in the discussion of papers. For one year, as an experiment, we shall not appoint any men to discuss the papers which are read by out-of-state guests. Sometimes the discussion is well done, and sometimes it is not. The local doctor who has not seen the paper is always at a disadvantage if he is obliged to say something extemporaneously about what has been said. We are going to limit the discussion to questions, which may either be asked orally or may be written on slips of paper, and the out-of-state guest is going to answer questions and do the talking.

For trustee, in place of Dr. Harriman, who has become trustee emeritus, the House of Delegates elected Dr. Samuel T. Ladd, of Portsmouth. Your new president is Dr. Clarence O. Coburn, of Manchester, and your new vice-president is Dr. James B. Woodman, of Franklin.

PRESIDENT LADD: We shall adjourn until two o'clock this afternoon.

WEDNESDAY AFTERNOON, MAY 18

The concluding session convened at two o'clock in the auditorium.

The program for the afternoon was as follows:

BLOOD DYSCRASIAS. J. H. J. Upham, Columbus, Ohio; president, American Medical Association.

DIAGNOSIS AND TREATMENT OF INTRA-ORAL CANCER. Hayes E. Martin, New York City. Discussion opened by George C. Wilkins, Manchester.

TREATMENT OF ARTHRITIS. Walter Bauer, Boston. Discussion opened by Ezra A. Jones, Manchester, and Lawrence R. Hazzard, Portsmouth.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24291

PRESENTATION OF CASE

First Admission. A twenty-two-year-old, native, male entered the hospital complaining of a chronic cough and blood-tinged sputum.

Several weeks before entry he first coughed up a small amount of blood-tinged sputum. He had been well previously, but for several years had been accustomed to raising a small amount of whitish sputum about once a day. During the month prior to entry he had noticed a little, sharp twinge of pain in the left chest on moving around in bed. Because of scoliosis he preferred to sleep on his left side. He had occasional night sweats, but had taken his temperature every day during the past month and it had always been normal. During the week prior to entry he was observed daily by his physician who found a constantly normal temperature. Four sputum examinations were negative for tubercle bacilli.

His past history revealed that at eight years of age his right leg and hip were injured in an automobile accident, with retardation of growth of the right lower limb and resultant scoliosis. His family history was negative for tuberculosis; his mother had died at the age of twenty-six of abdominal cancer. He was an only child.

Physical examination showed a well-developed and nourished young man with marked mid-dorsal kyphoscoliosis with rotation and posterior prominence of the right chest and compensatory lordosis secondary to an old hip injury. Slight dullness and poor aeration were noted in the left apex, but no rales were heard. Bronchoscopy showed the trachea to be badly deviated to the right and a slit-like opening of the left main bronchus, with evidence of external pressure. The abdomen was negative. The blood pressure was 110 systolic, 75 diastolic. No lymphadenopathy was noted.

The temperature was 98.6°F., the pulse 100. The respirations were 25.

The urine examination was negative. Examination of the blood showed a red-cell count of 5,400,000, and a hemoglobin of 80 per cent. The white-cell count was 14,000 with 86 per cent polymorphonuclears, 7 per cent small lymphocytes, 5 per cent large mononuclears and 2 per cent eosinophils. Several sputum examinations were negative for tubercle bacilli, and one was negative for blood. A tuberculin test with a 1:10,000 dilution showed a 7 mm. area of erythema in twenty-four hours. A blood Hinton test was negative.

X-rays showed a sharply defined homogeneous mass measuring 10 cm. in diameter in the upper anterior aspect of the left chest extending to the sternum in front. The esophagus passed behind it and the trachea to the right. There was no calcification within it. The remainder of the chest was negative.

The patient was given a total of 1400 r of x-ray therapy to the left upper chest and was discharged to return in three weeks for observation.

Second Admission (fourteen months later). After leaving the hospital the patient continued to cough up about a tablespoonful of green sputum a day. With a head cold two months after discharge blood tingeing of the sputum recurred and the patient returned to the x-ray department for treatment. Films showed no definite change in the appearance of the chest since the first observation. The dosage was raised to 800 r front and back, and four weeks later the mass had definitely reduced in size and density. The patient gained 10 lb., felt better and the blood tingeing ceased. Five months later he had chickenpox which was followed by a cold and recurrence of the blood-tinged sputum. This subsided spontaneously in about three days; but three months later, eleven months after his first entry to the hospital, this blood tingeing reappeared without other symptoms. X-rays showed a recurrence of the consolidation in the left upper lobe. At that time two definite cavities were noted in the area of density. A total of 2000 r of x-ray radiation were given over a period of three weeks and caused symptomatic improvement and marked reduction in the size of the mass and in the size of the cavities. One month later he acquired a cold and again began to cough up blood-streaked sputum daily. This persisted up to the time of entry, one month later. He had coughed up no frank blood and had lost no weight. His general condition was good.

Physical examination showed increased dullness in the left upper chest and high pitched breath sounds. The neck veins on the left were more prominent than normal. Blood examination showed a white-cell count of 5400 with 81 per cent polymorphonuclears, 1 per cent small lymphocytes, 12 per cent monocytes, 5 per cent eosinophils and 1 per cent basophils. The red-cell count was 4,240,000 with 75 per cent hemoglobin. Examination of twenty sputum specimens showed rare

spirochetes and various gram-positive cocci and bacilli, but no tubercle bacilli.

X-rays showed an extensive process in the left upper chest with cavitation and a new process extending out from the right hilus.

The patient remained in the hospital for three weeks and received 4300 r of x-ray therapy. The size of the lesion and of the cavity within it diminished, and he improved symptomatically.

Final Admission (twenty months later). During the interval the patient ran a variable course. His condition depended largely on the x-ray treatments that he received. He was given a total dosage of 25,500 r in amounts varying between 1000 and 4000 r for each series of treatments. Each consisted of 200 or 300 r given daily for a period of from four days to two weeks. The results were checked by frequent x-ray plates. Six months after discharge his chest was almost entirely clear, more normal than it had ever been during the period of observation. The patient was almost symptom free. Three months later consolidation with cavitation had reappeared in the middle of the right lung. He was coughing a good deal and had frequent night sweats. His general condition, however, remained good, and he tolerated the periodic treatments very well. Fourteen months after discharge an x-ray showed partial destruction and pathologic fracture of the third cervical vertebra with widening of the joint space between the third and fourth vertebrae. There was some kyphosis, but no definite displacement. The surrounding soft tissues were only slightly thickened. About four months later he began to run a downhill course. He developed pain in his neck, became weak and dyspneic, and began losing a good deal of weight. His cough increased, and he raised moderate amounts of mucopurulent sputum.

Physical examination showed a markedly emaciated adult man, breathing rapidly and appearing chronically and acutely ill. The posterior chain of cervical lymph nodes was moderately enlarged, hard, rubbery, non-tender and confluent. The chest showed dullness to flatness on both sides and coarse to medium crackling and moist rales with increased voice sounds over both apices. The heart beat was rapid and weak but regular; a rough systolic murmur was heard at the apex; A_2 was louder than P_2. The abdominal examination revealed a palpable spleen at the costal margin. The temperature was 98.8°F., the pulse 120. The respirations were 25. The blood showed a red-cell count of 4,200,000 with 80 per cent hemoglobin and a white-cell count of 13,400 with 95 per cent polymorphonuclears, 2 per cent lymphocytes and 3 per cent large mononuclears. X-rays showed multiple nodules in the lower parts of both lungs, with cavitation.

The patient failed rapidly. The chest became almost completely consolidated, and he was aroused with difficulty. His temperature varied from 96.4 to 99°F., pulse from 80 to 120, and respirations from 20 to 30. On the tenth hospital day, shortly after taking a drink of water, the nurse reported that respirations had ceased.

Differential Diagnosis

Dr. William B. Breed: I should like to list four conditions and discuss each one briefly: (1) lymphoma; (2) some mechanical difficulty on the basis of scoliosis which, with obstruction to the bronchus, would produce multiple abscesses; (3) bronchiogenic carcinoma; and (4) mycotic abscess of the lung.

From a therapeutic point of view this case has all the earmarks of a lymphoma. The two outs about it are the blood-tinged sputum and the development of cavities during its course. This man did very well on a lymphoma dosage of x-ray over a period of two years, and his improvement, both symptomatic and objective, depended entirely upon x-ray treatments. I have not seen any cases of lymphoma of the mediastinum which produced blood-tinged sputum or cavitation; therefore, I have to turn to the mechanical situation in his chest for some explanation of this symptom and this sign.

Bronchiogenic carcinoma would account for the blood-tinged sputum. It would account perhaps for the x-ray picture, with cavities behind an obstructed bronchus; but certainly the amount of x-ray therapy that was exhibited would have had no appreciable effect on its course. Furthermore, one would expect the story to be one with a constant downhill course and no real subjective benefit derived from the amount of x-ray therapy he received. On that basis I am almost willing to rule out bronchiogenic carcinoma.

In regard to mycotic abscess, I should think that we might have found something in the sputum during the course of twenty examinations. I am not at all clear as to the effect of x-ray therapy on mycotic abscess. On the record alone tuberculosis seems to have been pretty well ruled out. He had no fever, and no tubercle bacilli were discovered; moreover, I think that the x-ray does not suggest a tuberculous process.

I am going to ask Dr. Hampton to comment on the plates and also give some information about the possible results of x-ray therapy in the various conditions I have mentioned.

Dr. Aubrey O. Hampton: I do not know how many films were taken of this patient's chest

but I should guess around thirty or forty. I have picked out a few of the most interesting ones. As you see, he is so deformed it is difficult to tell which side is which. This is the left side, and this the area of density described in the left upper lobe. It is difficult to tell whether that shadow is in the apex of the upper lobe or in the mediastinum. Without bronchoscopy I do not believe I should have been able to get up enough courage to treat him. We did have one bronchoscopy, however, that showed external pressure on the left main bronchus so we began treating him, and the improvement in the patient was quite definite. After his first few treatments, he felt better. At his next visit some time later there were definite cavities in the solid area, but because of the previous improvement following radiation we believed he should have more.

DR. BREED: From the bronchoscopy you inferred the lesion was not in the left apex but probably in the mediastinum?

DR. HAMPTON: The first time, yes; but when he came in the second time we thought the process must be in the lung, because of the cavities. Believing that radiation was indicated we gave him 1000 r both on the front and back of the chest. He came in two months later showing definite improvement. The cavities had disappeared, and the area of density had reduced in size. The patient was working. His response was quite remarkable. He came back a short time later with cavities in both lungs and with a history of blood-tinged sputum. So we gave him 3900 r at that time, having to treat both sides, and suspecting that more than the lymphoma dose was indicated. We felt that the fractional doses we had given him before might have missed the lesion because of the marked deformity and the difficulty in centering the tube. At no time did he show an erythema of the skin because we did not treat the same area enough to produce an 800-r effect. He kept on working in spite of his illness. He was manager of a store.

This film was taken at the time of his next-to-last examination, and these nodules at both bases have appeared. This large cavity also had recurred in the right lung, and I think that we can demonstrate now why the bronchoscopist thought the left main bronchus was pressed upon. The deformity and apparent displacement were apparently due to the scoliosis. I do not believe his bronchus was definitely pressed upon at any time. These nodules are the first round masses that we saw in his chest which did not contain cavities.

DR. BREED: It is perfectly obvious that you started to treat this man for lymphoma. It also says in the record that each series consisted of 200 or 300 r daily and that you used a total of 4000 r for each series of treatment. That is higher than the lymphoma dose. Did you speed up the dosage?

DR. HAMPTON: We did. Four thousand roentgens is at least twice the lymphoma dose. We found we had to use twice the lymphoma dose before we could prolong the period improvement; then after a while we reached the point where we hoped we could collapse the cavities and keep them closed by heavier radiation.

DR. BREED: What is the effect of x-ray therapy on ordinary lung abscesses which might have developed because of scoliosis and bronchial obstruction?

DR. HAMPTON: I do not know. It is said that x-ray treatment is good for lung abscess, but I have no good reason to believe such a statement and no experience to prove it.

DR. BREED: Have you ever seen lymphoma with cavity formation like this?

DR. HAMPTON: Yes, once.

DR. BREED: That is the thing of course that led me astray because I have believed that peribronchial lymphoma, even with involvement of the lung, did not produce cavities. Of course he might have had more of a tendency to develop cavities because of his scoliosis. Is that a fair statement?

DR. HAMPTON: I do not remember seeing a patient with scoliosis and lung abscess. I think that this pressure on the bronchus was not really pressure but a bending of the bronchus that gave the impression of pressure. The bronchus was wide open and there was no trapping of air.

DR. BREED: But if you have a lymphomatous lesion in or around the bronchus you might at some point have obstruction to produce abscess cavities below it?

DR. HAMPTON: Yes.

DR. BREED: In the presence of a bad deformity of the bronchial tree, let us say?

DR. HAMPTON: Yes; that sounds reasonable.

DR. BREED: So we may have to make a fundamental diagnosis of lymphoma, with the development of secondary abscesses behind the lesion due to scoliosis in the presence of lymphoma, rather than to say that these cavities were caused solely by a lymphomatous process. Does the lymphoma dose of x-ray break down any lymph nodes?

DR. HAMPTON: Not very often. The absence of a high fever was quite helpful in ruling out abscess.

DR. BREED: I agree; that is hard to ignore.

DR. HAMPTON: We were impressed by the lack of visible lymph nodes, and the absence of a high fever did not make us too comfortable. As you know from the record, we tried many times to rule out tuberculosis. I demonstrated these films

at our x-ray conference and was open to criticism for treating without an absolute diagnosis.

DR. BREED: Do you ordinarily have to raise the dosage in lymphoma as high as in this case to get response?

DR. HAMPTON: Some lymphomatous masses require 2400 r before they are destroyed.

DR. BREED: So you would not have to bring in another diagnosis to explain why you had to raise the dosage here?

DR. HAMPTON: No; we tried small doses at first, and as I said, it was difficult to deliver the dose where we hoped to put it.

DR. RICHARD J. CLARK: Did he not have fever from time to time?

DR. HAMPTON: I thought he had more than the record states but not so much as you would expect with lung abscess.

DR. BREED: He might have had some fever from lymphoma. How often do you find infiltration of the bone by lymphoma?

DR. HAMPTON: In from 15 to 30 per cent of the cases.

I meant to say that actinomycosis of the lung would be treated in about this way; reports from the Mayo Clinic contend that such doses are necessary and that they do produce cures.

DR. WALTER BAUER: You would not have treated this man the way you did if you thought he had simple pulmonary abscesses?

DR. HAMPTON: No; if I had not been able to see the marked improvement in the patient from time to time I should not have continued treatment.

DR. BREED: I think it is reasonably clear, largely because of his remarkable response to therapy, that this man had lymphoma. I do not know of any other lesion that could respond as consistently and as well as this did. Whether these are lymphomatous masses that have broken down or whether they are abscesses secondary to his deformity and a bending of the bronchial tree, I cannot say. When I started I was not very willing to call the whole thing lymphoma, because I was not aware that lymphomatous masses break down into cavities. In any case, my fundamental diagnosis is lymphoma.

DR. TRACY B. MALLORY: Are there any other comments? Dr. Clark, you saw this patient.

DR. CLARK: I have not much to add except that the changes following x-ray treatment were certainly very striking up until this last winter when he began to fail. From then on he did not respond very well to treatment.

DR. BREED: Dr. Brailey brings up the question I brought up. How do you explain the blood-tinged sputum? Can it be explained on lymphoma alone or lymphoma plus the mechanical difficulty with the trachea?

DR. BAUER: Once you get abscess formation bleeding might take place.

CLINICAL DIAGNOSES

Lymphoma of lung with diffuse dissemination.
Pathologic fracture of cervical spine.

DR. BREED'S DIAGNOSES

Mediastinal lymphoma.
Lung abscess formation (? cause).
Scoliosis.

ANATOMICAL DIAGNOSES

Lymphoblastoma, Hodgkin's type, involving lungs, mediastinum, retroperitoneal nodes, spleen, liver, adrenals and pericardium.
Hydrothorax, bilateral.
Scoliosis.
Aplastic anemia.
Syringomyelia.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The autopsy showed that this man suffered from Hodgkin's disease, and so far as we could make out, from Hodgkin's disease alone. He had multiple nodules throughout the lungs; some of these were solid and others had central cavities. The solid nodules showed a caseous necrosis which in gross was practically indistinguishable from that of tuberculosis, but the sections show microscopically nothing whatever to suggest tuberculosis. The involvement was generalized. He had massive tumor in the retroperitoneal region, and a small nodule in the spleen; very little, however, in the liver. The spine was examined and we were unable to find a pathologic fracture or for that matter any evidence of Hodgkin's disease, either in the cervical region or in sections from the lumbar vertebrae. The marrow was, however, most abnormal, showing an almost total aplasia in all the areas examined. The tremendous amount of x-ray treatment may well have been responsible for this. A totally unexpected finding, which had apparently produced no symptoms whatever, was a severe grade of syringomyelia.

A PHYSICIAN: Did you examine the third cervical vertebra?

DR. MALLORY: Yes, we sawed through it and could find nothing in it grossly or microscopically.

CASE 24292

Presentation of Case

A twenty-eight-year-old, white, American housewife entered the hospital with the complaint of vomiting and abdominal pain of three weeks' duration.

She was well and healthy until three weeks before entry when, while eating her breakfast, she suddenly became nauseated and vomited. Shortly thereafter she had a sharp, bilateral, low-abdominal pain, radiating up to the umbilicus, which partially subsided after a brief period. For the succeeding three weeks she vomited almost everything she ate and only occasionally could keep down a meal. Her low-abdominal pain persisted, diminishing at times but never disappearing. She also began to have diarrhea, passing watery feces four or five times a day. The feces were light brown and did not contain blood. During her illness she also noticed gradual blurring of vision so that at entry she was unable to read print. One week before entry she began to have severe headaches in the frontal region, radiating to the top of the head. She also had several spells of chilly sensation accompanied by perspiration. She had no nocturia, dysuria or other urinary symptoms; and no dyspnea, orthopnea, palpitation, chest pain or edema of the face or ankles. She had no twitchings or convulsions, and no preceding sore throat or other upper respiratory infection. She had had catarrhal jaundice at the age of eight but had not had scarlet fever, rheumatic fever or repeated sore throats. She had never had any gynecological symptoms, and had had one uneventful pregnancy.

Physical examination revealed a well-developed and nourished and obviously ill woman with dry skin and tongue and pale mucous membranes. The heart was somewhat enlarged to the left, with a loud apical first sound. The aortic second sound was louder than the pulmonic. The blood pressure was 240 systolic, 170 diastolic. The lungs were clear. Some deep tenderness in the abdomen, without spasm, was noted. There was also some tenderness in the cul-de-sac and right vault on pelvic examination. The fundi showed bilateral papilledema, tortuous vessels, arteriovenous nicking, hemorrhages and exudate.

The temperature was 99.5°F., the pulse 110. The respirations were 20.

The urine had a specific gravity of 1.020, contained a trace of albumin, and showed 200 red cells, 5 white cells and occasional granular and hyaline casts per high-power field in the sediment. The blood showed a red-cell count of 3,470,000 with 75 per cent hemoglobin, and a white-cell count of 10,800 with 76 per cent polymorphonuclears. One stool gave a 4+ guaiac test, and three others were negative. Two blood Hinton and Wassermann tests were positive. The nonprotein nitrogen of the blood serum was 43, the calcium 10.44, the phosphorous was 3.64, and the total cholesterol 14.6 mg. per cent. The serum protein was 6.4 gm. per cent. The chlorides were equivalent to 99 cc. of N/10 sodium chloride, and the carbon-dioxide combining power was 60.6 vol. per cent. A phenolsulfonphthalein test of renal function gave less than 10 per cent excretion in two hours.

An x-ray of the chest showed marked increase in the size of the heart, particularly in the region of the left ventricle. A flat film of the abdomen showed unusually small kidneys, and an x-ray of the skull was negative except for two small areas of calcification in the right posteroparietal region close to the surface. An electrocardiogram showed rather low T waves, suggesting an abnormal myocardial state, and moderate left-axis deviation.

She remained in the hospital four weeks, during which time the red-cell count fell to 2,800,000, and the blood protein to 5.6 gm. per cent, and the nonprotein nitrogen rose to a terminal level of 265 mg. per cent. The urine continued to contain large amounts of albumin, although fewer red cells and casts were seen in the sediment. The temperature fluctuated between 98.6 and 101, rising terminally to 102.5°F.

On the day after entry she developed acute pulmonary edema while being given an intravenous injection of dextrose in saline. This cleared up rapidly, and for the first week she showed some improvement. Her diarrhea and vomiting were less severe, and she was able to eat small amounts of food. During the second week she developed some pruritus and hiccough and began to vomit about once daily. On the twenty-first day she developed a very loud pericardial friction rub, which could be heard and felt all over the left chest, and a few fine rales at the left base, and on the twenty-seventh day began to show muscle twitching. She died on the twenty-ninth day.

Differential Diagnosis

Dr. Richard J. Clark: I presume that the areas of calcification noted in the skull plate are probably small blood vessels and of no significance.

At the onset we may say with reasonable certainty that this patient had renal disease and died with uremia. That is fairly easy. The next question which comes to mind is, Is this an acute process or one of long standing? We have no history of any previous nephritis. We are told that she had one uneventful pregnancy, but we do not know when that was; presumably she had a fairly normally functioning heart and kidneys at that time, for there were no complications. In other

words this process is probably one of relatively short duration, possibly a matter of six or seven years at the outside, since she is twenty-eight years old. There are certain things, however, that make us feel it is a chronic process: first, the extremely high blood pressure — one rarely sees a pressure of that degree in acute nephritis; secondly, the severe changes and tortuosity of the vessels of the fundi; and thirdly, the fairly high degree of cardiac hypertrophy. The x-ray findings of the abdomen show unusually small kidneys, which is again fairly strong evidence in favor of a rather severe, long-standing process. It is somewhat surprising that the patient had no urinary symptoms, such as nocturia, previous to this acute onset, but that in itself does not rule out the presence of a latent, chronic nephritis.

The urinary picture at the time of entry is surprising, particularly the notation of a specific gravity of 1.020. However, it is only a solitary report and in the same specimen 200 red cells were found in the sediment. This again is a somewhat surprising finding in chronic nephritis. With that number of red cells we might wonder whether the patient had an acute hemorrhagic nephritis, or whether there was something else going on. We do know that in some cases of chronic glomerulonephritis there are periods when showers of large numbers of red cells are found, possibly from rupture of one of the small vessels in the calices. It is possible that the large number of red cells may be a factor in causing an apparent rise in the specific gravity of the urine. The fact that the patient had such a low phenolsulfonphthalein test certainly indicates a fairly severe degree of renal incompetence, and I am inclined to throw out the specific gravity of 1.020. Again we come back to the question of whether there may be an element of acute nephritis engrafted upon a chronic nephritis. There is no history of oliguria, and none of the things that we might expect in acute nephritis. Then, the question comes up whether this patient had malignant hypertension with a superimposed acute nephritis. This I think is unlikely. The age of the patient, the severe degree of hypertension, the cardiac findings and the retinal findings, all fit in perfectly well with malignant hypertension, but it is unusual to get very severely contracted kidneys in a relatively short period of time simply from malignant hypertension, and this is perhaps the strongest reason of all for throwing out the diagnosis.

We now come to the positive Hinton and Wassermann tests. We wonder if they could be related to the main picture. There is no record of any antiluetic treatment to serve as a kidney irritant. The only type of Bright's disease produced by syphilis is the nephrotic variety, which the patient quite obviously did not have. In regard to another possible association with syphilis, one wonders if there was any chance of the patient's having amyloid kidneys. In very rare instances they are found in syphilis of long duration; they may be contracted and may be associated with hypertension and uremia, but this is unusual, and in the absence of any long-standing debility and in the absence of other evidence of amyloid disease, such as large liver and spleen, I think we can throw out the diagnosis. Some people believe that malignant hypertension may be associated with syphilis as a causal factor, but the evidence for this is quite slim. So I am going to say that syphilis is a side issue and has nothing to do with the main picture.

Then to return to the onset of the present illness, we find a somewhat perplexing situation. The patient had an acute gastrointestinal upset, which was followed by lower abdominal pain. I think we can say that this is probably not an acute surgical lesion. We do know that severe gastrointestinal episodes may well be the first symptoms in the onset of renal failure and are not infrequently seen in severe chronic glomerulonephritis. Also this same group of gastrointestinal symptoms is occasionally associated with malignant hypertension. If we are going to say that these symptoms are secondary to uremia, we are perhaps a little surprised to find at entrance that she had a nonprotein nitrogen of only 43 mg. per cent and none of the chemical findings that we should ordinarily associate with a severe degree of uremia. I suppose it is possible to say that she had had a higher nonprotein nitrogen, but I doubt such a sequence. I am going to attribute these findings to the onset of the uremia rather than to secondary changes. It is possible that we might find some ulcers, possibly of the uremic type, in the lower bowel. We also have to think of the possibility of a small thrombosis in the lower mesentery. We have a 4+ guaiac test on one occasion, which might be associated with either of these situations, as could the rather indefinitely described tenderness in the lower abdomen and pelvis. It is not unusual for patients who are coming down with severe nephritis or uremia to be operated on with erroneous diagnoses of various acute surgical lesions.

I believe that we should make one main diagnosis in this case, namely chronic glomerulonephritis, with very severely contracted kidneys. I believe that the patient had a very markedly hypertrophied heart of the hypertensive type, probably with some dilatation and a terminal pericarditis of the uremic type, and probably a terminal bron-

chopneumonia. There may be low colonic ulceration, or possibly mesenteric thromboses.

Clinical Diagnoses

Malignant hypertension.
Hypertensive heart disease.
Chronic vascular nephritis.
Uremia.
Latent syphilis.

Dr. Clark's Diagnoses

Chronic glomerulonephritis.
Hypertrophy of the heart, hypertensive type.
Uremic pericarditis.
Bronchopneumonia.
Uremic enteritis?
Mesenteric thrombosis?
Syphilis (serological).

Anatomical Diagnoses

Nephritis, chronic vascular, malignant type.
Cardiac hypertrophy, left ventricular.
Pericarditis, acute, with effusion.
Enteritis, acute.

Pathological Discussion

Dr. Tracy B. Mallory: The kidneys in this patient were distinctly small, but not quite so small as the x-ray report would indicate, since they weighed 200 gm. The usual chronic vascular nephritis or its more malignant counterpart does not produce a great deal of atrophy in the kidneys, but kidneys weighing down to 200 gm. are perfectly consistent with that diagnosis. On histological examination the lesion was a typical, malignant, vascular nephritis. There was nothing to suggest any underlying lesion, although I cannot rule out the possibility that at some long distant period she might have had glomerulonephritis. The vascular changes were very diffuse, involving many organs. There was also a diffuse enteritis of the terminal ileum without any sharp border, disappearing gradually as one went upward — about the sort of thing we frequently see in terminal uremia and call uremic enteritis. It can occur in either the small or large bowel. There were no sharply delimited necrotic lesions. The heart, as expected, was hypertrophied, — weighing 350 gm., which for a very small woman should be definite hypertrophy, — but there were no other findings of significance. I frankly do not know whether syphilis played any role in her symptomatology. Some people believe it is of significance as an etiologic agent in malignant hypertension. The evidence, however, is far from convincing. We found nothing at autopsy which would justify an anatomical diagnosis of syphilis.

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The Journal does not hold itself responsible for statements made by any contributor.

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SURVEY ON THE ADEQUACY OF MEDICAL CARE

The national survey on the adequacy of medical care now being made by the American Medical Association, though a bit belated, is one of the most important events for the medical profession that has occurred in a number of years. The best answers to any criticisms that have been directed against organized medicine will be found in this report, provided the doctors and medical societies are willing to co-operate in making the undertaking a success. If this survey is half-heartedly done and later developments result in protests on the part of doctors because of governmental interference in the conduct of their lives and practice, there will be but little justification for complaint.

There are several reasons why a survey of this sort should be conducted by the organized forces of the medical profession. In the first place, physicians are more likely to determine the true state of the need, supply and quality of medical care than any other group of individuals. Secondly, if inadequacies exist, — and there can be no doubt but that they do, — those who are practicing medicine should be vitally interested in the correction of these defects. And, thirdly, if medicine is to be criticized, — as it has been and as it will be, — the profession should have at hand factual data with which to refute unfair criticism.

During the collection of these data there should be no letup in the attempts to correct the inadequacies of medical care that are known to exist. President Frothingham emphasized this point in his report "On the State of the Society" (New Eng. J. Med. *219*: 1-3, 1938). He also deplored the lack of interest shown by the district societies in organizing health-service councils, the object of which essentially concerns this same problem — the adequacy of medical care.

It is to be hoped that in the present instance all members of the district societies will do their utmost to co-operate in filling out the necessary forms at the earliest possible moment. If situations arise where specific information is required concerning local situations, a letter to Dr. Rosco G. Leland, Bureau of Economics, American Medical Association, will receive prompt attention and an immediate reply.

SEX CRIME

Man usually tries to find a general rule which he can apply to all problems. This has been a constant search in medicine. At the summit in therapeutics we find general rules such as: quinine for malaria, antitoxin for diphtheria, liver for pernicious anemia, and a few others. Yet, on the whole, medicine has stood for treatment of the patient as an individual.

The rule of society from time immemorial has been to punish all criminals. During the last generation the principle of individual treatment has been applied in criminology, and the medical profession has had a great deal to do with establishing this principle. Yet, when the public is

aroused by a series of revolting sex crimes, once again the demand is for extreme punishment as a remedy.

It seems wise to refer to sex crime as a social problem because this term refers to acts all the way along from those which are better thought of as little sins to those which are the most atrocious deeds with which we have to deal. There is a medical component in all this, but the social factors are so overwhelming in importance as to overshadow the medical. One of the oldest of crimes, adultery, is rarely punished, and more often involves moral turpitude rather than mental peculiarity. The more serious crimes of incest and rape usually call for medical advice. When we come to those crimes involving definitely abnormal traits we are well within the realm of psychiatry, and at least on the diagnostic side, the doctor has much to contribute.

In the recent, widely publicized cases, medical knowledge appeared to have been adequate and medical advice was evidently sound but the administration of the procedures set up by society to deal with these situations was faulty. Evidently the law is adequate and the doctors can be trusted to do their duty, but we must insist on better administration.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

CASE HISTORY NO. 81. CENTRAL PLACENTA PREVIA

Mrs. D. S., a twenty-nine-year-old secundipara, was admitted to the hospital on May 12, 1938, at term, because of vaginal hemorrhage. Early in the morning of admission, she was awakened from her sleep because of a vaginal hemorrhage which soaked through two sheets. She had no abdominal pain or cramps. She was advised over the telephone to enter the hospital immediately.

Her family history was negative. The patient had always been well. Her tonsils and adenoids were removed in her childhood. She had had one normal full-term delivery, with a normal convalescence, five years previously. Her menstrual periods were established at the age of fourteen and had always been regular, occurring every thirty days and lasting three or four days; there were no clots or dysmenorrhea. Her last menstrual period was August 5, 1937, and her expected date of confinement was May 12, 1938.

Her present pregnancy had been uneventful until the onset of the hemorrhage. Her blood pressure and urine had been normal.

Physical examination revealed a well-nourished, small-statured woman. The mucous membranes were slightly pale. The heart and lungs were normal. The temperature was 98°F., the pulse 90, and the respirations 20. She was flowing moderately. Abdominal examination revealed a full-term pregnancy, breech presentation. The fetal heart could be heard faintly on the right at the level of the umbilicus. There were no uterine contractions, and the abdomen was soft.

The patient and her husband were typed and cross-matched for a possible transfusion. Urinalysis was negative. The operating room was prepared for operation. Under aseptic conditions a speculum was inserted into the vagina; no cervical polyp or ruptured varix was found. There was a steady stream of blood coming from the cervical os. A diagnosis of central placenta previa was made, and cesarean section was decided upon.

Under nitrous-oxide, oxygen and ether anesthesia, a low transverse cervical section was performed, and a female child weighing 6 lb., 11 oz., was delivered by the breech and cried readily. The placenta was on the posterior wall and covered the os completely. It was removed intact with the membranes complete. Bleeding was not excessive. The uterine incision was sutured in two layers with No. 2 chromic catgut, the first interrupted and the second a continuous inverting stitch. The vesicouterine flap of peritoneum was sutured to the uterus with a continuous stitch of No. 0 chromic catgut, making the uterine incision retrovesical. The abdomen was then closed in layers. The mother's condition at the end of the operation was good, and she did not need to be transfused.

Her convalescence was uneventful. Her postoperative hemoglobin was 60 per cent (Sahli), and she was given ferrous sulfate by mouth to remedy this condition. The sutures were removed on the ninth postoperative day; the incision was clean and well healed. She was out of bed on the twelfth day and discharged on the fourteenth. Her hemoglobin on discharge was 75 per cent, and the baby was over birth weight, weighing 6 lb., 15 oz.

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

Comment. Placenta previa at term is characterized by painless bleeding. The diagnosis is usually made after a vaginal examination, which is the only way that the condition can be accurately differentiated from partial separation of the placenta. In this case, the amount of bleeding coming through the cervix at the time of inspection was probably so great that it was felt that an exact diagnosis obtained by palpation of the placenta through the cervix was unnecessary. The actual bleeding made immediate operation necessary, and in the absence of labor with no dilatation, cesarean section was the operation of choice. Had this patient been in labor with an obliterated cervix dilated to two or three fingerbreadths the case might have been bagged extra- or intra-ovularly, — in either case the baby would have been despaired of, — or a Braxton-Hicks maneuver might have been performed — in which case the baby's chances of survival would also have been negligible.

Before operation is attempted in cases of this sort the patient should be typed and a donor obtained so that transfusion can be done immediately if necessary. If indicated, the patient should be transfused before operation.

Operation itself in this case revealed a placenta which entirely overlay the cervix and confirmed the preoperative diagnosis of complete placenta previa. There is a definite trend toward a test of labor in subsequent pregnancies when a cesarean section has been done for any condition other than disproportion. Some men feel that the often accepted axiom, "Once a cesarean, always a cesarean," is not necessarily true. When the low sections were first advocated, it was believed that the danger of rupture of the scar was less than in the classical section. It may be less, but ruptures have occurred in subsequent labors. Therefore each case should be given definite consideration, and if it is decided that any patient who has had a cesarean should be allowed a test of labor, it must be known that the condition of the cervix is very favorable for a normal delivery and that there is no possible chance of difficulty in labor from relative disproportion, and the labor must be carefully watched by the obstetrician, so that he may be ready, at a moment's notice, to enter the abdomen if there are any signs of rupture.

DEATHS

CUMMINGS — Alvah C. Cummings, M.D., of Newton, died July 11. He was in his sixty-fifth year.

Dr. Cummings was a graduate of Dartmouth College and of Harvard Medical School. He held memberships in the Massachusetts Medical Society, the American Medical Association, the Rotary Club and the Elks and belonged to the Masonic Order. He had practiced in Newton for forty years and had been city physician of Newton for four years.

His widow and daughter survive him.

TALAMO — Haskell Talamo, M.D., of Worcester, died July 10. He was in his forty-second year.

Born in Worcester, he graduated from English High School in 1913, from Clark University in 1916 and from Harvard Medical School in 1926. Dr. Talamo received a fellowship from Cornell University Medical College in 1928 and was appointed to the staff of the New York Nursery and Child's Hospital where he served under Dr. Oscar M. Schloss.

A fellow of the Massachusetts Medical Society, he was a member of the Worcester District Medical Society. Dr. Talamo also held memberships in the American Medical Association and the American Academy of Pediatrics. He was on the staffs of Memorial Hospital and Fairlawn Hospital, Worcester, and the Children's Hospital, Boston, and was on the consulting staffs of several other hospitals.

His widow, father, mother and two brothers survive him.

NOTICES

SOUTH END MEDICAL CLUB

The South End Medical Club will visit the Boston Sanatorium, 249 River Street, Mattapan, on Tuesday, July 26, at 10 a. m. An interesting program will be presented by members of the staff. From there the Club will go to the Prendergast Preventorium of the Boston Tuberculosis Association, 1000 Harvard Street, Mattapan, where Dr. Eli Friedman will demonstrate the different skin tests for tuberculosis, and where lunch will be served.

On Tuesday, August 2, the Club will visit the East Concord Street Health Unit at 10 a. m. All the facilities for the diagnosis, management and treatment of tuberculosis will be inspected, including the Pneumothorax Clinic. From there the Club will visit the Sheltered Workshop of the Boston Tuberculosis Association, 35 Tyler Street, Boston, for inspection and lunch.

Those interested are invited to make these visits with the Club.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, July 25

Tuesday, July 26
*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

Friday, July 29
*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

Saturday, July 30
*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Robert T. Monroe.

*Open to the medical profession.

July 26 and August 2 — South End Medical Club. See notice above.

September 12-14 — American Association for the Study of Goiter. Page 545, issue of March 24.

September 12-15 — American Congress of Physical Therapy. Page 946, issue of June 2.

October 8 and November 15 — American Board of Ophthalmology. Page 282, issue of February 10.

October 17-21 — Clinical Congress of the American College of Surgeons, New York City.

October 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C.

March 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

District Medical Societies

HAMPDEN

Meeting will be held on the fourth Tuesday in July.